INDUSTRIAL MEDICINE *and* SURGERY

Established 1932

feature issue . . . .

McINTYRE RESEARCH FOUNDATION CONFERENCE

Silicosis and other Industrial Pulmonary Diseases

JUNE 1960

Volume 29 Number 6

Full-Time Corrective Action

in mild depression and chronic fatigue

Deaner®

tablets deanol acetamidobenzoate

Improves *night-time* restoration and *day-time* performance

- Gradually prepares patient to awaken better rested and more alert
  ...permits sounder sleep
  ...lessens sleep requirements
- Increases daytime energy
- Counteracts mild depression
  ...acts to stabilize emotionally disturbed patients with or without concomitant disease
- Useful in treating children with learning defects and behavior problems...lengthens attention span
- Unlike monoamine inhibitors. It is not necessary to monitor Deaner's administration with repeated laboratory tests...Deaner may be given with safety to patients with previous or current liver disease, kidney disease or infectious diseases.

'Deaner' is supplied in scored tablets containing 25 mg. of 2-dimethylaminoethanol as the *p*-acetamidobenzoic acid salt.

In Mild Depression

chronic fatigue and many other emotional and behavioral problems

Literature, file card and bibliography on request

Riker Northridge, California

for all steroid-responsive dermatoses

Rx METI-DERM® CREAM

prednisolone topical

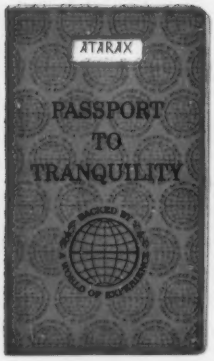
Schering

also METI-DERM Ointment with Neomycin when secondary infection is a factor, too

5-474

A GUIDE TO

THE REALMS OF INDUSTRIAL PRACTICE BEST ATTAINED WITH



ATARAX®

(brand of hydroxyzine)

World-wide record of effectiveness—over 200 laboratory and clinical papers from 14 countries. **Widest latitude of safety and flexibility**—no serious adverse clinical reaction ever documented. **Chemically distinct among tranquilizers**—not a phenothiazine or a meprobamate. **Added frontiers of usefulness**—antihistaminic; mildly antiarrhythmic; does not stimulate gastric secretion.

Areas of Special Usefulness	Supportive Clinical Observation	...and for additional evidence
IN AMBULATORY NEUROTICS	"Since this drug does not impair psychic function... it is especially well suited for ambulatory neurotics who must work, drive a car, or operate machinery." Ayd, F. J., Jr.: New York J. Med. **57**:1742 (May 15) 1957.	Garber, R. C., Jr.: J. Florida M. A. **45**:549 (Nov.) 1958. Menger, H. C.: New York J. Med. **58**:1684 (May 15) 1958.
IN PREMENSTRUAL TENSION	"The consensus appears to be that this agent has a definite calming effect on nervous and agitated subjects with symptoms such as... premenstrual tension...." Shalowitz, M.: Geriatrics **11**:312 (July) 1956.	Schuller, E.: Gaz. des Hôpitaux **129**:391 (Apr. 10) 1957. Dolan, C. M.: California Med. **88**:443 (June) 1958.
IN ALCOHOLISM	Atarax "...is the tranquilizer of choice in the treatment of both acute and chronic stages, inpatient and outpatient, because of its safety, versatility, flexibility, and effectiveness." McGettigan, D. L.: In press.	Cohen, S.: Am. Pract. & Digest Treat. **8**:946 (June) 1957. Garber, R. C., Jr.: J. Florida M. A. **45**:549 (Nov.) 1958.

Dosage: One 25 mg. tablet or one tbsp. syrup q.i.d. For emotional emergencies, 25-50 mg. (1-12 cc.) I.M., 3-4 times daily q. 4 h. **Supplied:** Tiny 10 mg., 25 mg., and 100 mg. tablets. Syrup (10 mg. per tsp.), pint bottles. Parenteral Solution: 25 mg./cc. in 10 cc. multiple-dose vials; 50 mg./cc. in 2 cc. ampules. Prescription only.

Rx PFIZER

New York 17, N. Y.
Division, Chas. Pfizer & Co., Inc.
Science for the World's Well-Being

INDUSTRIAL MEDICINE and SURGERY

ESTABLISHED 1932

The International Journal of Medicine in Industry

JUNE, 1960
VOLUME XXIX
NUMBER 6

CONTENTS

Introduction 231
W. B. Dix and J. W. G. Hannon, M.D.

A Practical Method of Providing a Suitable In-Plant Environment 233
K. E. Robinson

The Clinical Significance of Cavitation in Anthracosilicosis 238
Peter A. Theodos, M.D.

Diffuse Lung Lesions Which Mimic Pneumoconioses 243
H. S. Van Ordstrand, M.D.

The Value of Large Lung Sections in the Study of Diseases of the Chest 247
William Taylor, M.D.

Johannesburg Pneumoconiosis Conference 251
A. D. Campbell

Problems of Silicosis in British Columbia 260
E. O. T. Simpson

Recent Trends in Silicosis among Miners in Ontario 265
W. C. Wheeler and A. H. Sellers, M.D.

Mortality from Occupational Diseases of the Lung in Ontario 273
A. H. Sellers, M.D.

The Dust Control and Ventilation Services of the Quebec Metal Mines Accident Prevention Association 278
Gerard Grassmück

Emphysema in Relation to Occupation 283
Professor J. Gough

Roentgen Study of Silicosis in Ceramic Workers Who Have Received McIntyre Aluminum Powder 286
J. W. G. Hannon, M.D., Paul G. Bovard, M.D., Leslie R. Osmond, M.D.

Practical Evaluation of the Function of the Lungs 290
George R. Meneely, M.D.

The 1960 Industrial Health Conference 305

Books 307

Editorial: The Thirteenth International Congress on Occupational Health 308

Personal News & Notes 4, 6

Comment and Opinion 35

Index of Advertisers 44

INDUSTRIAL MEDICINE AND SURGERY, The Journal of Medicine in Industry (with which are consolidated "The Industrial Doctor" and "International Journal of Medicine and Surgery"): Entered as second class matter August 28, 1941, at the Post Office at Chicago, Illinois, under the Act of March 3, 1879. Reentered, October 3, 1949. Additional entry at Sheboygan, Wisconsin. Published on the fifth of each month by INDUSTRIAL MEDICINE PUBLISHING COMPANY, Miami, Florida. STEPHEN G. HALOS, Chairman; STEPHEN F. HALOS, President; HAROLD A. HOOPER, Treasurer; CAREY P. MCCORD, M.D., Editor; JOHN A. LAWRENCE, Managing Editor; A. D. CLOUD, Editorial Consultant; LUCILLE H. GROSS, Circulation Manager; T. W. PINKARD, Production Manager. EXECUTIVE OFFICES, 400 S.W. 69th Avenue, Miami 44, Florida. PUBLICATION, EDITORIAL AND CIRCULATION matters should be addressed to P. O. Box 44-306, Miami 44, Florida.

Subscriptions in United States $6.00 per year, $10.00 for 2 years; in Canada $8.00 per year, $14.00 for 2 years; in other countries $12.00 (U.S. Currency) per year, $20.00 for 2 years. Single copies $1.25. Title Registered in United States Patent Office. Registered Canada, 1942.

Copyright, 1960, by INDUSTRIAL MEDICINE PUBLISHING COMPANY.

CHANGE OF ADDRESS: Five weeks' notice required; changes cannot be made without old address as well as new; please furnish zone number for new address if city requires it.

BPA

PERSONAL NEWS AND NOTES

CHARLES G. MERCKEL, M.D., has been appointed Medical Director for Mountain View Operations of Sylvania Electric Products Inc. He replaces JOHN LOPES, M.D., who served Sylvania on a part time basis and has returned to private practice.

DR. MERCKEL was formerly on the medical staff of the Southern California Edison Company. He also was preferred medical examiner for the Mutual Life Insurance Company of New York and on the staffs of the Hospital of the Good Samaritan and the Los Angeles County General Hospital before joining Sylvania.

In 1933 DR. MERCKEL received the A.B. degree from Wayne University in Detroit, Michigan, and later was awarded the M.S. degree on the basis of experimental work in the physiology of reproduction. In 1940 he was awarded the M.D. degree from Wayne University School of Medicine. He is the author of several papers published in medical journals and for six years was an instructor at the University of Southern California School of Medicine. He is a specialist in internal medicine.

DR. MERCKEL saw four years of active duty with the U. S. Navy during World War II and now holds the rank of commander in the U. S. Naval Reserve.

He is a member of several medical societies including the Los Angeles County Medical Association, the California State Medical Association, the American Medical Association and the Industrial Medical Association.

WILLARD R. DAVIES, M.D., a consultant in chest diseases to four Nassau County hospitals, has been appointed an Expert Consultant on Dust Diseases to the New York State Workmen's Compensation Board by COL. S. E. SENIOR, Board Chairman.

DR. DAVIES is consultant in medicine, diseases of the chest and allergy, at Nassau Hospital, Mineola; at Mercy Hospital, Rockville Centre; at Meadowbrook Hospital, Hempstead; and at Southern Nassau Communities Hospital, Oceanside.

He has specialized in diseases of the chest and allergy since 1932. He was an intern and Resident Associate Superintendent of the Nassau County Sanatorium between 1928 and 1932, and also engaged in the chest survey of Nassau County school children in 1928 and 1929. He was graduated from the Medical School of the University of Pennsylvania in 1927.

He is a member of the American Medical Association, the Medical Society of the State of New York, the Nassau County Medical Society, the American College of Physicians, a founding member of the American College of Chest Physicians, the President-Elect of the New York State Chapter of the American College of Chest Physicians, a member of the American Trudeau Society and of the Trudeau Society of Eastern States, and of the New York Society of Internal Medicine. He also is a founding member of the Nassau Internists Society, and a member of the International Society of Internal Medicine and of the New York Academy of Sciences. He is the author of many professional papers on chest diseases.

FRANK H. DE GRACE, M.D., was recently elected President of the Industrial Medical Association of New Jersey. He is chief physician of Wright Aeronautical Division, Wood-Ridge.

DR. DE GRACE joined Wright's in 1951 as plant physician and a year later was promoted to his present post. A graduate of St. John's University and Loyola University, he served internships at St. Michael's Hospital, Newark, and St. Mary's Hospital, Passaic.

He served as an Army medical officer during World War II and was on active duty at Pearl Harbor on December 7, 1941. He was discharged from service with the rank of major.

PAUL J. WHITAKER, M.D., Medical Director for the Allis-Chalmers Manufacturing Co., Milwaukee, Wisconsin, has been

★ AMERICAN ASSOCIATION OF RAILWAY SURGEONS ★

Officers — 1960-1961

President:
WALTER D. ABBOTT, M.D., Des Moines, Iowa.
President-Elect:
CHESTER C. GUY, M.D., Chicago, Ill.
Vice Presidents:
WILLIAM H. SAXTON, M.D. Huron, So. Dak.
RUFUS B. ROBINS, M.D., Camden, Arkansas
W. K. WEST, M.D., Oklahoma City, Okla.
Treasurer:
JOHN R. WINSTON, M.D., Chicago, Ill.
Secretary:
CARL Y. WERELIUS, M.D., Chicago, Ill.
Recorder:
JAMES K. STACK, M.D., Chicago, Ill.

Executive Board

ARTHUR R. METZ, M.D., Chicago, Ill., Chairman
ROBERT M. GRAHAM, M.D., Chicago, Ill.
RAYMOND HOUSEHOLDER, M.D., Chicago, Ill.
A. M. W. HURSH, M.D., Philadelphia, Pa.
J. M. L. JENSEN, M.D., Chicago, Ill.
RAYMOND B. KEPNER, M.D., Chicago, Ill.
ERNEST C. OLSON, M.D., Chicago, Ill.
RAY S. WESTLINE, M.D., Chicago, Ill.
T. L. HANSEN, M.D., Chicago, Ill.

Asst. to Secretary-Treasurer:
MR. FRANK PARKER, 59 E. Van Buren St., Chicago, 5, Ill.

"an ideal adjuvant
to open therapy of BURNS . . ."[1]

BETADINE™ AEROSOL SPRAY

(ACTIVE INGREDIENT: POVIDONE IODINE*)

provides iodine's microbicidal activity, yet will not sting, burn, irritate; minimizes the risk of iodine sensitization even on denuded skin areas.[1]

BETADINE AEROSOL SPRAY in burn therapy — *prevents infection, has unexcelled topical pathogenicidal activity; it avoids the discomfort and necessity of large dressings; forms a bacteria-free eschar aiding epithelization; helps reduce the loss of electrolytes and body fluids; eliminates noxious odors common to burn wounds; Betadine Aerosol Spray also aids in skin transfer procedures.*

HOW TO USE: Betadine Aerosol Spray should be applied with minimum delay over the entire injured area. Apply twice daily for first three days, and once daily thereafter until separation of the eschar begins. **INDICATIONS:** In "prepping" procedures Betadine Aerosol Spray eliminates the hazard of microbial colony transfers, minimizes destruction of tissue, helps maintain sterile field throughout operation, covers body crevices, and saves time. **SUPPLIED:** Betadine Aerosol Spray: 3-oz. bottle. **ALSO AVAILABLE:** Betadine Antiseptic Solution: 8-oz., 16-oz., and 1-gallon bottles.

Topical pathogenicide in acute and chronic skin infections, effective against antibiotic resistant organisms, including staph. aureus.

TAILBY-NASON COMPANY, INC. Dover, Delaware

Pat. 2,739,922 G A AF. Corp.

1. Garnes, A.L.; et al: Am. J. Surg. 97:49, 1959.

named Secretary of the American Academy of Occupational Medicine for 1960.

D. JOHN LAUER, M.D., Past President (1959-1960) of the Industrial Medical Association, and former Medical Director of Jones-Laughlin Steel Corporation, Pittsburgh, Pennsylvania, has been named Medical Director of International Telephone and Telegraph Company, New York.

LEO WADE, M.D., Medical Director of Esso Standard Oil Company, New York City, has been named President of the American Delegation to the 13th International Congress on Occupational Health, which is to be held in New York City, July 25 - 29, 1960. Please see announcement below for information.

SEWARD MILLER, M.D., Director, Institute of Industrial Health, University of Michigan, Ann Arbor, has been named Chairman of the United States Delegation to the 13th International Congress on Occupational Health.

SARA WAGNER, R.N., Director of Nursing Service, Standard Oil Company of New Jersey, has been named Secretary of the United States Delegation to the 13th International Congress on Occupational Health.

13th International Congress on Occupational Health
New York City, July 25-July 29, 1960

An International Congress on Occupational Health is held every three years under the auspices of the Permanent Committee and International Association on Occupational Health. A committee in the country where the meeting is held takes responsibility for organizing the Congress. In the United States in 1960, the Organizing Committee is headed by LEO WADE, M.D., 15 West 51st Street, New York City.

The scientific program is as follows: Monday — Plenary Session, Influence of Environmental Factors, Medical Practices, Social and Legal Aspects, Environmental Hygiene, *Work Physiology and Psychology.

Tuesday—Environmental Hygiene, Medical Practices, Social and Legal Aspects. Influence of Environmental Factors, *Administrative Practices, Specific Industries, Surgical Practices, Influence of Environmental Factors, Work Physiology and Psychology, *Education and Training.

Wednesday — Environmental Hygiene, Medical Practices, Administrative Practices. Influence of Environmental Factors (Pneumoconiosis), Field Trip.

Thursday — Specific Industries, Work Physiology and Psychology, Administrative Practices, Influence of Environmental Factors, *Medical Practices, Environmental Hygiene, Surgical Practices, Education and Training, Work Physiology and Psychology, *Influence of Environmental Factors (Radiation).

Friday — General, Environmental Hygiene, Medical Practices, Influence of Environmental Factors, *Social and Legal Aspects. Plenary Session.

Commercial exhibits of products of interest to physicians in occupational health practice are being arranged. Scientific exhibits of general interest will also be shown.

The American Express Company has been designated as the official travel agency of the Congress. Prospective members of the Congress will find this company helpful in arranging details and answering questions about travel.

Tours to various points of interest will be arranged under the charge of the American Express Company. These tours will include toxicology research laboratories, industrial plants of various kinds, governmental health and industrial hygiene institutions, educational centers for industrial health. American Express will provide details about these tours.

Several events are being planned for general participation of members of the Congress. General entertainment events and trips for the ladies will also be available. Details of ladies' events may be obtained later from the local American Express agent.

The Waldorf-Astoria Hotel has been selected as the headquarters and all meetings and functions will be held there. Rooms for registrants at the meeting are being held at the following hotels. For other housing facilities local American Express agents should be consulted.

	Rates Per Day		
	Single	Two Persons Double and Twin Beds	Suite
Waldorf Astoria*	$10-$15	16-21	35-50
Belmont Plaza	9.85	13.85	—
Lexington	9.65	14.90	—
Shelton Towers	9-18	14-22	—
Beverly	—	—	28-32

*Headquarters Hotel

Room reservations should be made as early as possible. Please indicate the expected date of arrival and expected date of departure from the hotel of your choice. When possible, it will be found most convenient to be housed in the headquarters hotel. Foreign members attending the conference may wish to share a room with a friend to reduce expenses. If this is desired, please make these personal arrangements in advance and indicate the person sharing the room on the reservation form. *If a room reserved in advance is not used on the first day, it can be made available for others.*

Apply for room reservations to R. E. ECKARDT, M.D., Secretary General, P. O. Box 45, Linden, New Jersey, U. S. A., by May 1, 1960.

See the editorial on page 308 of this issue.

*Major Sessions — simultaneous translation available.

KEEP THEM ON THE JOB WITH BUFFERIN®

Fast relief of headache and muscle-joint pains without salicylate gastric irritation

8.7% of employees to whom you give plain aspirin may return the same day with an upset stomach.* This aspirin intolerance apparently is due to the local action of aspirin.[1]

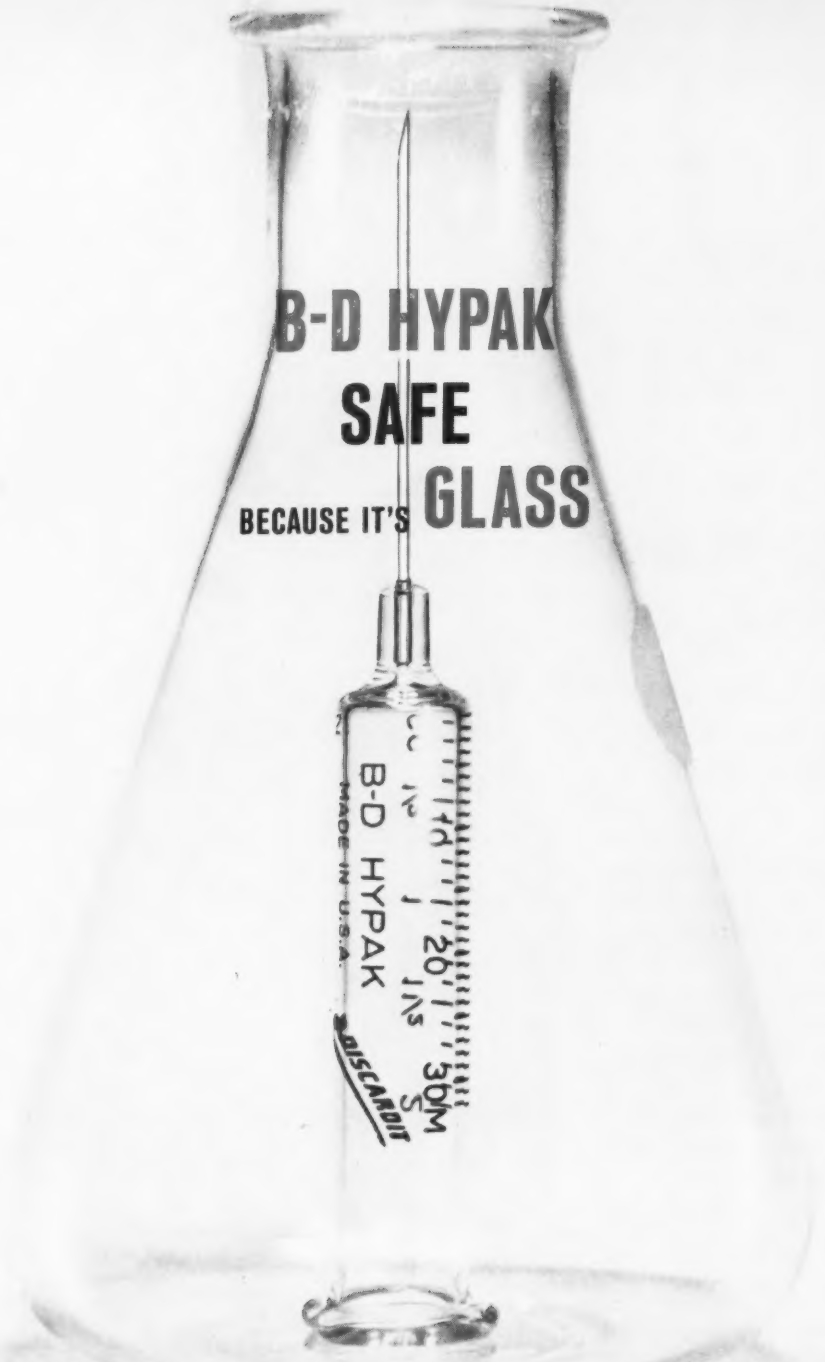
BUFFERIN with its antacid components sharply reduces the incidence of gastric intolerance while enhancing the comfort of the patient.

For fast analgesic and anti-inflammatory benefits without salicylate gastric irritation, dispense BUFFERIN.

*Based on 8.7% patient intolerance to salicylate therapy reported by Tebrock, H. E.: Ind. Med. & Surg. *20*:480-482, 1951.

1. Kelly, J. J., Jr.: Am. J. Med. Sci. *232*:119-128 (Aug.) 1956.

Bristol-Myers Company, 630 Fifth Avenue, New York 20, New York



SAFE FOR TODAY'S MEDICATIONS...AND TOMORROW'S

NO CAUTION LABEL NEEDED—Use it with **any** injectable medication...there is no danger of solvent action on the barrel. **SAFE**—B-D Control guarantees sterility, nontoxicity, non-pyrogenicity. **ECONOMICAL**—Disposability eliminates time-consuming, pre-use preparation. **PRECISE**—Exclusive tip design reduces medication loss.

B-D **BECTON, DICKINSON AND COMPANY · RUTHERFORD, NEW JERSEY**

a B-D DISCARDIT product

B-D HYPAK AND DISCARDIT ARE TRADEMARKS OF BECTON, DICKINSON AND COMPANY, INC. 79060

NOW–for more comprehensive control of

'pain & spasm'

- pain due to or associated with
- spasm of skeletal muscle

a NEW Robins *muscle relaxant-analgesic*

Robaxisal®

ROBAXIN® WITH ASPIRIN

ROBAXISAL, a new dual-acting muscle relaxant-analgesic, effectively treats both skeletal muscle spasm and severe pain due to or associated with the spasm. Each Tablet contains:

- A relaxant component — Robaxin* — widely recognized for its prompt, long-lasting relief of painful skeletal muscle spasm, with unusual freedom from undesired side effects . . . . . 400 mg.

  *Methocarbamol 'Robins' U.S. Pat. No. 2770649.

- An analgesic component—aspirin—whose pain-relieving effect is markedly enhanced by Robaxin, and which has added value as an anti-inflammatory and anti-rheumatic agent . . . . (5 gr.) 325 mg.

INDICATIONS: ROBAXISAL is indicated when analgesic as well as relaxant action is desired in the treatment of skeletal muscle spasm and severe concurrent pain. Typical conditions are disorders of the back, whiplash and other traumatic injuries, myositis, and pain and spasm associated with arthritis.

SUPPLY: ROBAXISAL Tablets (pink-and-white, laminated) in bottles of 100 and 500.
Also available: ROBAXIN Injectable, 1.0 Gm. in 10-cc ampul. ROBAXIN Tablets, 0.5 Gm. (white, scored) in bottles of 50 and 500.

A. H. ROBINS CO., INC., Richmond 20, Va.

Making today's medicines with integrity . . . seeking tomorrow's with persistence

Why Clinical Judgment Often Dictates Altafur for Peroral, Systemic Therapy of Pyodermas

Gratifying Therapeutic Response

ALTAFUR was found "highly satisfactory in most of the primary and secondary bacterial dermatoses treated to date," including "pyodermas . . . caused by antibiotic resistant strains of staphylococci."[1] In a nationwide survey[2] there were **94%** satisfactory results (cured or improved) among 159 patients treated with ALTAFUR for pyodermas.

Virtually Uniform in vitro Susceptibility of Staphylococcus aureus

99.5% of isolates (214 of 215) from patients with staphylococcal infections—including many antibiotic-resistant strains—proved sensitive in vitro to ALTAFUR in tests conducted across the nation.[3] **99.7%** of staphylococcal isolates (334 of 335) at a large general hospital—including many antibiotic-resistant strains—proved sensitive in vitro to ALTAFUR.[4]

Wide, Stable Antimicrobial Spectrum

"Because of its relationship to previously developed nitrofurans, it is anticipated that [ALTAFUR] will retain its original spectrum after longstanding clinical usage."[5] Development of significant bacterial resistance to ALTAFUR has not been encountered to date.[6]

Minimal Side Effects

Side effects are easily avoided or minimized by these simple precautions: 1) *alcohol should not be ingested in any form, medicinal or beverage, during* ALTAFUR *therapy and for one week thereafter* 2) each dose should be taken with or just after meals, and with food or milk at bedtime (to reduce the likelihood of occasional nausea and emesis).

1. Weiner, A. L.: Paper presented at the Conference on Recent Advances in the Treatment of Chronic Dermatoses, University of Cincinnati (Ohio), Nov. 5, 1959. 2. Compiled by the Medical Department, Eaton Laboratories, from case histories received. 3. Christenson, P. J., and Tracy, C. H.: Current Therapeutic Research 2:22, 1960. 4. Glas, W. W., and Britt, E. M.: Proceedings of the Detroit Symposium on Antibacterial Therapy, Michigan and Wayne County Academies of General Practice, Detroit, Sept. 12, 1959, p. 14. 5. Leming, B. H., Jr.: Ibid., p. 22. 6. Investigators' reports to the Medical Department, Eaton Laboratories.

Tablets of 250 mg. (adult)

and 50 mg. (pediatric)

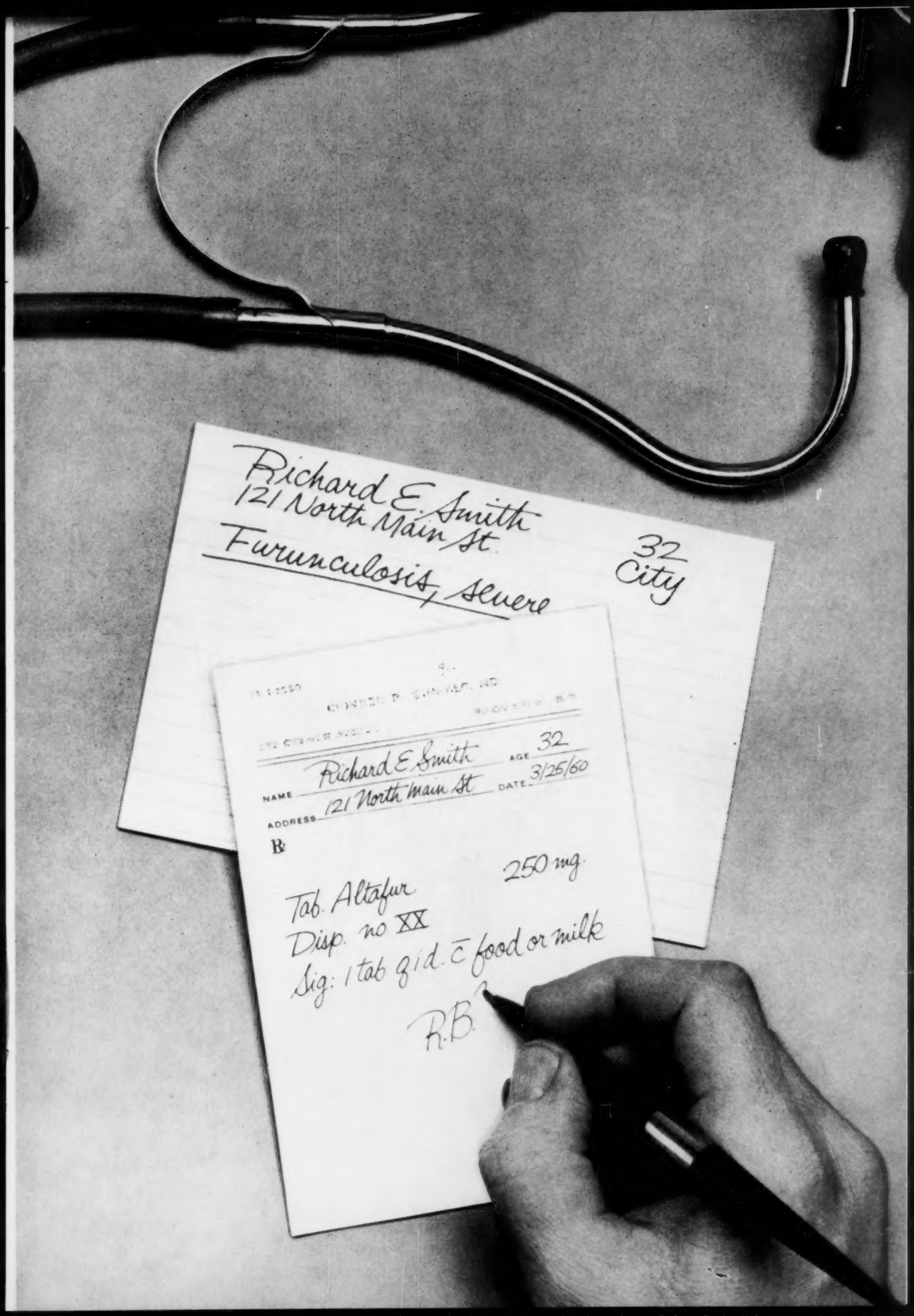
bottles of 20 and 100

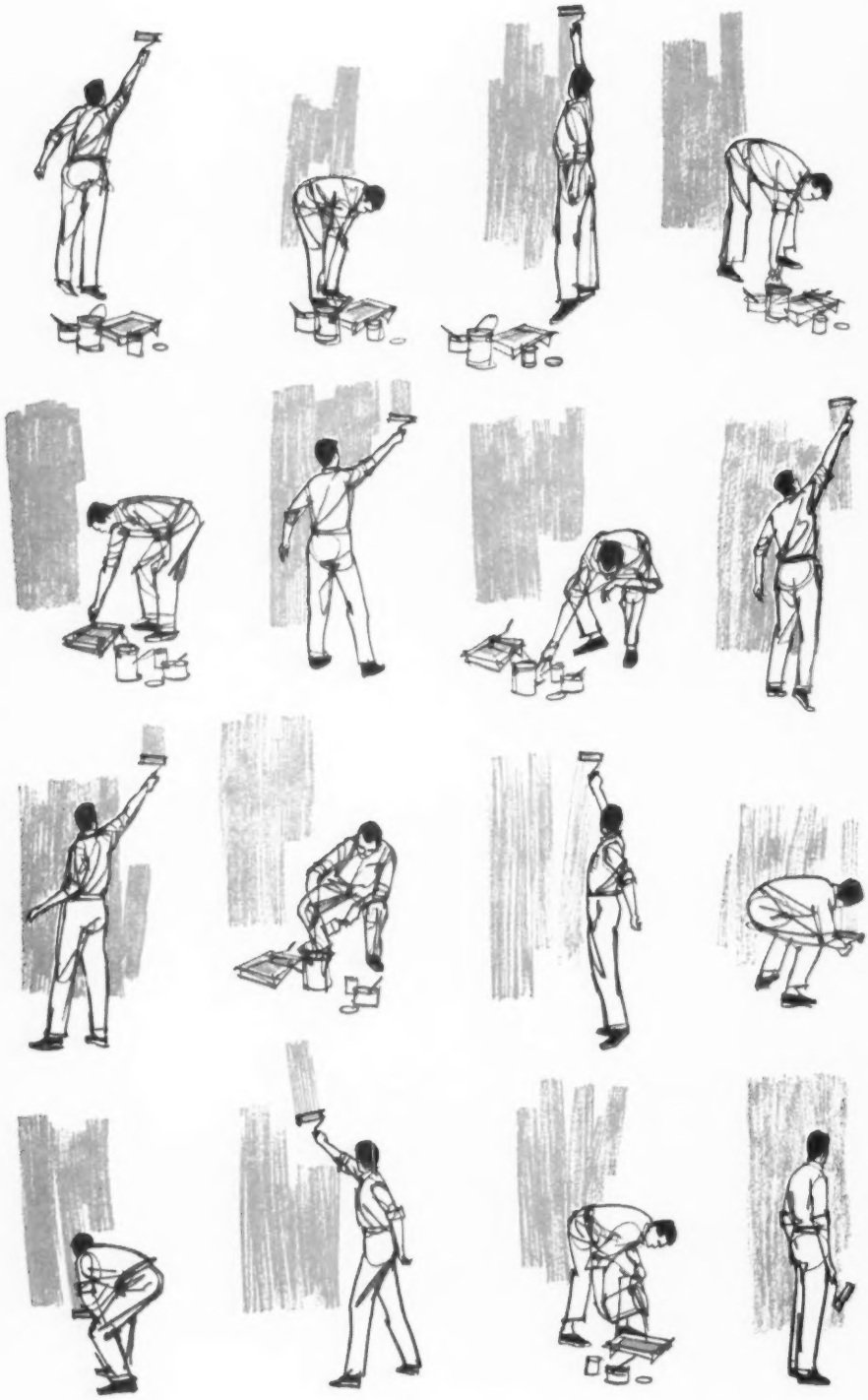
Altafur®

brand of furaltadone

NITROFURANS . . . a unique class of antimicrobials

EATON LABORATORIES, NORWICH, NEW YORK


Richard E. Smith
121 North Main St.
Furunculosis, severe
32
City
NAME Richard E. Smith AGE 32
ADDRESS 121 North main St DATE 3/25/60
℞
250 mg.
Tab. Altafur
Disp. no XX
Sig: 1 tab q i.d. c̄ food or milk
R.B.



when that early Monday morning telephone call is from a weekend do-it-yourselfer

"...and this morning, Doctor, my back is so stiff and sore I can hardly move."

now...there is a way to prompt, dependable relief of back distress

the pain goes while *the muscle relaxes*

POTENT – rapid relief in acute conditions

SAFE – for prolonged use in chronic conditions

notable safety – extremely low toxicity; no known contraindications; side effects are rare; drowsiness may occur, usually at higher dosages

rapid action, sustained effect – starts to act quickly, relief lasts up to 6 hours

easy to use – usual adult dosage is one 350 mg. tablet 3 times daily and at bedtime

supplied – as 350 mg., white, coated tablets, bottles of 50; *also available for pediatric use:* 250 mg., orange capsules, bottles of 50

WALLACE LABORATORIES, New Brunswick, New Jersey

SOMA™

(CARISOPRODOL WALLACE)

Armour Pharmaceutical Company Announces a New Systemic Enzyme

Chymoral

I AM PLEASED to inform you of the latest development of our Company's research.

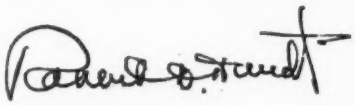
To the expanding field of systemic anti-inflammatory enzymes we are introducing Chymoral. It is a specially coated tablet specifically designed for intestinal absorption. The activity is supplied by a purified concentration which has specific trypsin and chymotrypsin activity in a ratio of approximately six to one.

During past months, clinical investigators have evaluated Chymoral in a wide range of inflammatory conditions. They have reported to us as well as to the medical journals on the therapeutic response, convenience and safety of this oral form.

Patients have responded very well on a Chymoral dosage schedule of 2 tablets q.i.d. and one tablet q.i.d. for maintenance. Important, too, is the fact that where other therapeutic agents were used there were no incompatibilities.

Chymoral is indicated in a wide range of inflammatory conditions to control inflammation, curtail swelling and curb pain.

If you would care to review some of the published reports on Chymoral we shall be happy to send reprints of these papers to you.



Robert A. Hardt
President

1. Beck, C.; Levine, A. J.; Davis, O. F., and Horwitz, B.: Clinical Studies with an Oral Anti-inflammatory Enzyme Preparation. Accepted for publication in Clin. Med. (March) 1960. 2. Billow, B. W.; Cabodevilla, A. M.; Stern, A.; Palm, A.; Robinson, M., and Paley, S. S.: Clinical Experiences with an Oral Anti-Inflammatory Enzyme for Intestinal Absorption. Accepted for publication in Southwestern Med. (May) 1960. 3. Teitel, L. H.; Siegel, S. J.; Tendler, J.; Reiser, P., and Harris, S. B.: Clinical Observations with Chymotrypsin in 306 Patients. Accepted for publication in Indust. Med. (April) 1960. 4. Clinical Reports to the Medical Dept., Armour Pharmaceutical Company, 1959. 5. Reich, W. J., and Nechtow, M. J.: Scientific Exhibit, Chicago Medical Society (March) 1960. 6. Taub, S. J.: Paper presented Annual Meeting, ΠΛΚ Medical Fraternity, Miami, Florida (March) 1960.

© 1960, A. P. Co.

another sinus headache on the assembly line?

No question about it! Each year, sinus or frontal headache causes costly worker inefficiency, tardiness, and absenteeism. **Sinutab,®** the specific medication for sinus or frontal headache, can help prevent many of these wasted man-hours. Sinutab aborts the pain, decongests to relieve pressure, and comforts the patient with mild tranquilizing action. A Sinutab patient remains a working patient. Sinutab in easy-to-swallow tablets is supplied in bottles of 30. Dosage: two tablets every four hours.

WARNER CHILCOTT
MORRIS PLAINS, N.J.

SIN-1P02

HYDELTRA® -T.B.A.

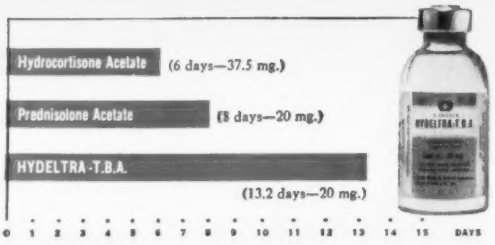
(Prednisolone *tertiary*-butylacetate, Merck)

for relief that lasts – longer

in *TENOSYNOVITIS*– often frees "locked" tendons without need for surgery

- Osteoarthritis
- Rheumatoid arthritis
- Acute gouty arthritis
- Bursitis
- Tendinitis
- Trigger finger
- Tenosynovitis
- Trigger points
- Tennis elbow
- Lumbosacral strain
- Capsulitis
- Frozen shoulder
- Coccydynia
- Rheumatoid nodules
- Fibrositis
- Tensor fascia lata syndrome
- Collateral ligament strains
- Sprains
- Radiculitis
- Osteochondritis
- Ganglia

Anti-inflammatory effect lasts longer than that provided by any other steroid ester


Hydrocortisone Acetate
(6 days—37.5 mg.)
Prednisolone Acetate
(8 days—20 mg.)
HYDELTRA-T.B.A.
(13.2 days—20 mg.)
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 DAYS

DOSAGE: the usual intra-articular, intra-bursal or soft tissue dose ranges from 20 to 30 mg. depending on location and extent of pathology.

SUPPLIED: Suspension 'HYDELTRA'-T.B.A. 20 mg./cc. of prednisolone *tertiary*-butylacetate, in 5-cc. vials.

MSD

MERCK SHARP & DOHME
DIVISION OF MERCK & CO., INC.
PHILADELPHIA 1, PA.

A Vacation from Hay Fever is a Real Vacation

ANYWHERE – ANYTIME

Just a "poof" of fine **NTZ** *spray brings relief* IN SECONDS, FOR HOURS

NTZ is a potentiated, balanced combination of these well known synergistic compounds:

Neo-Synephrine® HCl, 0.5%
– dependable vasoconstrictor and decongestant.

Thenfadil® HCl, 0.1%
– potent topical antihistaminic.

Zephiran® Cl, 1:5000
– antibacterial wetting agent and preservative.

NTZ® NASAL SPRAY

Supplied in leakproof, pocket size squeeze bottles of 20 cc.

Winthrop LABORATORIES New York 18, N. Y.

what lurks beyond the broad spectrum?

Broad spectrum antibiotics provide the best means of combating pathogenic organisms which range all the way from large protozoa through gram-negative and gram-positive bacteria to certain viruses at the far end of the spectrum.

But beyond the spectrum lurk pathogenic fungi. It is increasingly apparent that fungal superinfections may occur during or after a course of broad spectrum antibiotics.[1,2] Long term debilitating diseases, diabetes, pregnancy, corticosteroid therapy, high or prolonged antibiotic dosage, and other causes may predispose to fungal superinfections.[1,3,4]

Mysteclin-V controls infection and prevents superinfection. It makes a telling assault on bacterial infections and, in addition, prevents monilial overgrowth.[2,5-8] Mysteclin-V is a combination of tetracycline phosphate complex for reliable control of most infections encountered in daily practice, and Mycostatin, the safe antifungal antibiotic. When you prescribe Mysteclin-V, you provide "broad spectrum therapy" plus extra protection that extends beyond the spectrum of ordinary antibiotics.

'MYSTECLIN'®, 'SUMYCIN'®, 'MYCOSTATIN'®, AND 'FUNGIZONE'® ARE SQUIBB TRADEMARKS.

In pediatrics: Mysteclin-F for Aqueous Drops and Mysteclin-F for Syrup are phosphate-potentiated tetracycline combined with the new antifungal antibiotic, Fungizone (amphotericin B). They provide good-tasting, fruit-flavored aqueous liquids for your pediatric patients.

Supplied: Mysteclin-V Capsules (250 mg./250,000 u.); Half-strength Capsules (125 mg./125,000 u.); Mysteclin-F for Syrup (125 mg./25 mg. per 5 cc.); for Aqueous Drops (100 mg./20 mg. per cc.)

References: 1. Dowling, H. F.: Postgrad. Med. *23*:594 (June) 1958. 2. Gimble, A. I.; Shea, J. G., and Katz, S.: Antibiotics Annual 1955-1956 New York, Medical Encyclopedia Inc., 1956, p. 676. 3. Long, P. H., in Kneeland, Y., Jr., and Wortis, S. B.: Bull. New York Acad. Med. *33*:552 (Aug.) 1957. 4. Rein, C. R.; Lewis, L. A., and Dick, L. A.: Antibiotic Med. & Clin. Ther. *4*:771 (Dec.) 1957. 5. Stone, M. L., and Mersheimer, W. L.: Antibiotics Annual 1955-1956, New York, Medical Encyclopedia Inc., 1956, p. 862. 6. Campbell, E. A.; Prigot, A., and Dorsey, G. M.: Antibiotic Med. & Clin. Ther. *4*:817 (Dec.) 1957. 7. Chamberlain, C.; Burros, H. M., and Borromeo, V.: Antibiotic Med. & Clin. Ther. *5*:521 (Aug.) 1958. 8. From, P., and Alli, J. H.: Antibiotic Med. & Clin. Ther. *5*:639 (Nov.) 1958.

Mysteclin-V

Tetracycline Phosphate Complex (Sumycin) and Nystatin (Mycostatin)

SQUIBB *Squibb Quality—the Priceless Ingredient*

PROTEIN

essential for growth and development

With other nutrients, protein affects the differentiation of cells during development . . . participates in all metabolic processes which result in growth . . . and is stored as the structural part of all of the soft tissues of the body.

In relation to body weight, requirement for protein . . . expressed as such or as essential amino acids and total nitrogen . . . is highest during infancy . . . slowly decreasing as rate of growth decreases until adult size is reached. During pregnancy and lactation, when growth is supported by maternal tissues . . . protein requirement again increases.

Assuming an ideal pattern of amino acids in dietary protein . . . the changing minimum requirement has been expressed by the Food and Nutrition Board in gms. of protein per kilogram of body weight as follows . . . 1.76 from birth to 3 months . . . 1.21 from 3 to 6 months . . . 0.95 from 6 to 9 months . . . 0.77 from 9 to 12 months . . . 0.54 from 1 to 6 years . . . 0.50 from 7 to 9 years . . . 0.44 from 10 to 12 years . . . 0.42 from 13 to 15 years . . . 0.35-0.36 for girls and boys 16 to 19 years . . . 0.44 during the second half of pregnancy . . . and 0.56 during lactation with 850 ml. of milk secreted daily.

Assigning a biological value of 79 to cow's milk, the requirements change respectively to . . . 2.23, 1.53, 1.20, 0.97, 0.68, 0.63, 0.56, 0.53, 0.44 to 0.46, 0.56 and 0.71 grams of cow's milk protein required per kilogram of body weight . . . from birth until 20 years of age . . . and during pregnancy and lactation.

As increasing amounts of high quality protein are fed to children, the amino acid pattern of the diet decreases in importance. As long as the biological value of the protein of a diet falls above 60, the amino acid pattern is not critical . . . for amino acid requirements can readily be satisfied if enough protein is eaten.

Milk is man's first dietary source of protein . . . providing the total protein needs during the first months of life. One quart of milk daily can provide the pre-school child up to 85 percent of his protein allowance . . . and the teen-age youth one-third to one-half of his protein allowance.

The nutritional statements made in this advertisement have been reviewed by the Council on Foods and Nutrition of the American Medical Association and found consistent with current authoritative medical opinion.

Since 1915...promoting better health through nutrition research and education

NATIONAL DAIRY COUNCIL

A non-profit organization

111 NORTH CANAL STREET • CHICAGO 6, ILLINOIS

THIS ADVERTISEMENT IS ONE OF A SERIES. REPRINTS ARE AVAILABLE UPON REQUEST

for dry, red, scaly, cracked, soap-abused hands

acid-mantle®

CREME pH 4.2 – LOTION pH 4.5 DOME

The normal, protective acid mantle of the skin, which has been removed by washing with soap and detergents, is instantly restored and maintained by ACID MANTLE Creme and Lotion, and normal physiological function and appearance return to the skin.

Creme in 1 oz. tubes, 4 oz., 1 lb. and 5 lb. jars. Lotion in 4 oz. squeeze bottles, 1 pint, and ½ gallon

Sig.: Apply after wet work and after washing hands. Samples and literature on request.

Stops the Flame of Skin Inflammation . . .

domeboro® tabs pH 4.2

DOME

THE MODERN BUROW'S SOLUTION used all over the world . . . for contact dermatitis due to alkalis, chemicals, oils, soaps, plastics, etc. Also in powder packets.

DOME CHEMICALS INC. NEW YORK 23 • LOS ANGELES 46
In Canada: 2765 Bates Rd., Montreal

relieves musculoskeletal pain

Zactirin®

Ethoheptazine Citrate (75 mg.) with Acetylsalicylic Acid (325 mg.), Wyeth

NON-NARCOTIC

ANALGESIA PLUS

ANTI-INFLAMMATORY

BENEFITS

In an industrial clinic, ZACTIRIN provided effective analgesia for patients with painful musculoskeletal conditions resulting from sprains, low back pain, fractures of small bones, and trauma of contusions. ZACTIRIN provided analgesia "... equivalent in effectiveness to that of codeine, 30 mg., plus aspirin ... but the usually expected side effects of codeine were noticeably absent."*

The investigators concluded "... the effectiveness and lack of side effect liability permit us to keep many employees at work who formerly were sent home."*

Supplied: Tablets, bottles of 48.

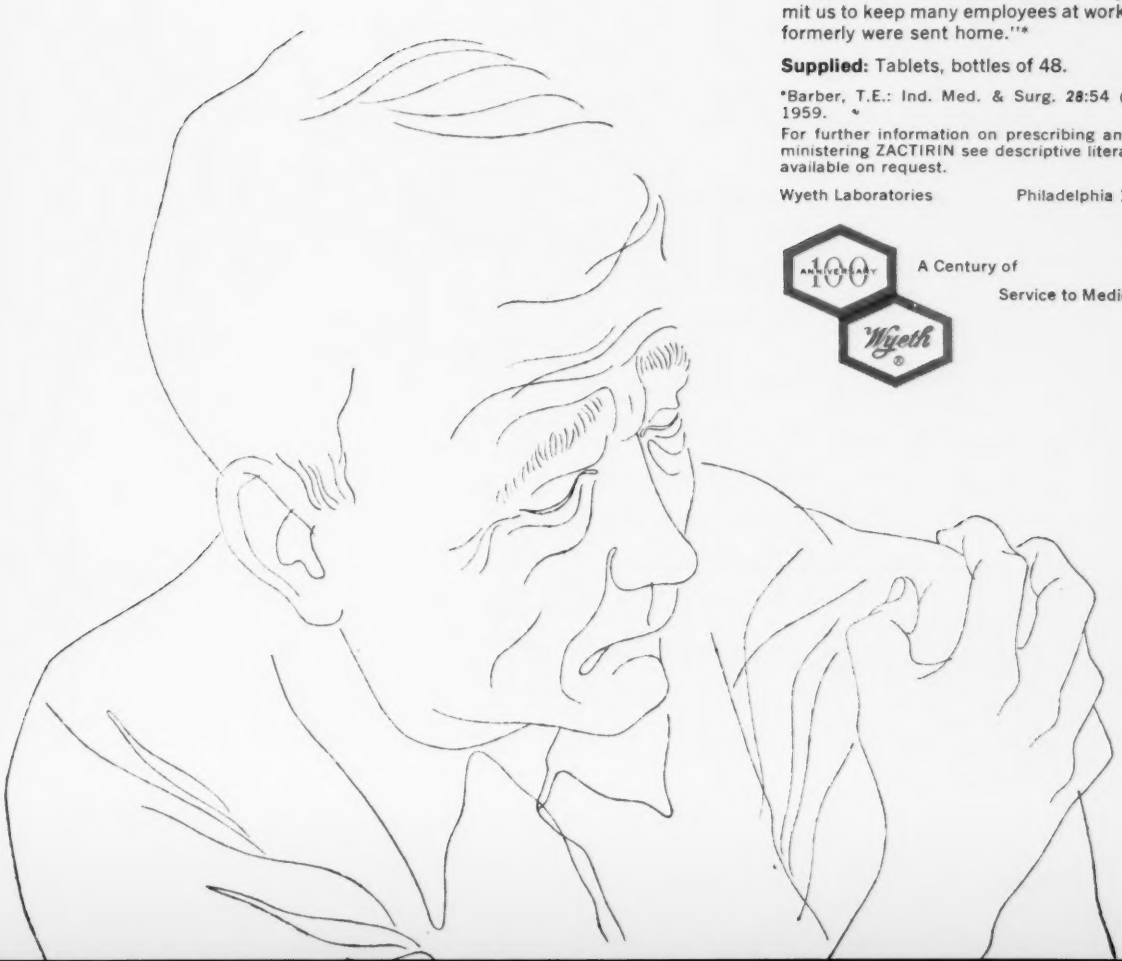
*Barber, T.E.: Ind. Med. & Surg. **28**:54 (Feb.) 1959.

For further information on prescribing and administering ZACTIRIN see descriptive literature, available on request.

Wyeth Laboratories Philadelphia 1, Pa.

100 ANNIVERSARY Wyeth®

A Century of Service to Medicine



To lick a stubborn industrial problem...

AQUA IVY AP® TABLETS

for desensitization to

POISON IVY and POISON OAK

For outdoor workers one of the most annoying problems is the danger of poison ivy and poison oak. For employers the problem means absenteeism for treatment...or prophylactic injections.

Now, however, by oral prophylaxis with AQUA IVY TABLETS poison ivy and poison oak among industrial workers can be a thing of the past. And here's why:

Extensive clinical tests show that AQUA IVY TABLETS:

...ARE EFFECTIVE —*good to excellent results in 95% of cases tested.*[1]

...GIVE LASTING IMMUNITY —*patch tests show immunity continues up to 12 months.*[2]

...ARE SAFE—*no sensitization, flare-up of existing lesions or gastro-intestinal irritation. Proved suitable for children.*[1-4]

Protection established before outdoor work begins

Because immunization procedures require a build-up period, AQUA IVY TABLETS are taken in advance of the season to be sure of protection when needed. Many experience benefits after 100 tablets sufficient to provide summer-long immunity. This requires about 8 weeks of tablet taking as directed. Others will require the dosage up to 200 tablets.

To assure your workers of protection against poison ivy and poison oak this season—put them on AQUA IVY TABLETS now! You'll see a big difference in absenteeism and in general outdoor working efficiency once the fear, pain and inconvenience of poison ivy or poison oak dermatitis is removed.

AVAILABLE: *bottles of 1,000, 300 and 100 tablets, supplied through usual drug channels.*

REFERENCES:

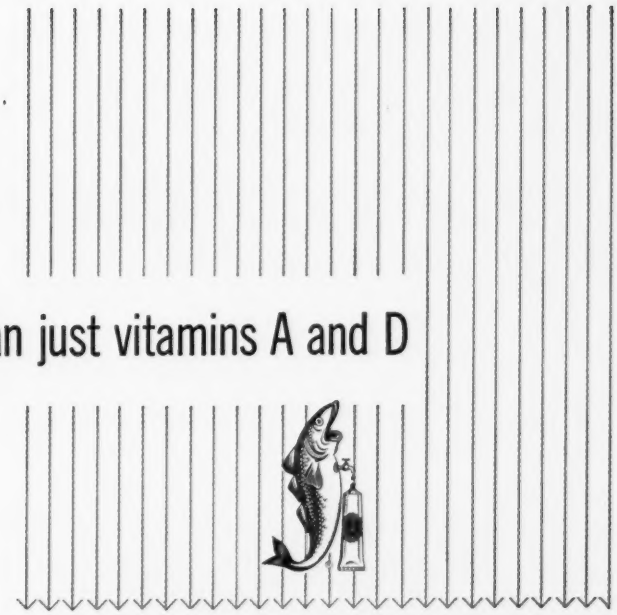
1. Langs, R. J. and Strauss, M. B.: J. Allergy. 30: 130-139 (March-April), 1959.
2. Langs, R. J., Fuchs, A. M., and Strauss, M. B.: Indus. Med., 28: 6, 257-261 (June); 1959.
3. Kligman, A. M.: J.A.M.A., 171: 592, (Oct. 3) 1959.
4. Personal Communications.

For free illustrated booklet "Facts You Should Know About Poison Ivy and Poison Oak" write Dept. IM

SYNTEX CHEMICAL COMPANY, INC.

10 East 40th Street, New York 16, N. Y.

more than just vitamins A and D



DESITIN® ointment

also provides
unsaturated fatty acids as well as the vitamins A and D (of high grade Norwegian cod liver oil)—essential to skin health and integrity

and ingredients that are emollient, lubricant, gently astringent, protective, and aid tissue repair (zinc oxide, talcum, petrolatum and lanolin)

in a smooth creamy ointment so processed that one application of Desitin soothes, protects, and promotes healing for hours in...

- **traumatic and infectious wounds**
- **burns (first, second, third degree)**
- **pressure sores and ulcers**
- **pilonidal cysts and sinuses**
- **ano-rectal wounds** • **chest wounds**

Samples Please write... **DESITIN CHEMICAL COMPANY**
812 Branch Avenue, Providence 4, R. I.

New ORAL DECONGESTANT

DRISTAN®

TABLETS

afford a new degree of symptomatic relief from

- **SINUSITIS**
- **THE COMMON COLD**
- **ALLERGIC DISORDERS**

Minimizes the Hazard of Drowsiness on the Job

as compared with other preparations containing antihistamines

Unique 3-layer decongestant tablet development offers a combination of ingredients never before possible to put into a single tablet.

DRISTAN—*Faster In Therapeutic Action*

Regular Enteric Coated Tablet	3-Layer Uncoated Dristan Tablet
Disintegrates in 3 hours	Disintegrates in 7 minutes

Dristan Decongestant Tablets—afford *a new degree of relief* from the symptoms of sinusitis, nasal allergies, postnasal drip and the common cold by releasing:

(1) A POWERFUL VASCOCONSTRICTOR . . . phenylephrine hydrochloride. This shrinks swollen sinus membranes and promotes proper drainage.

(2) A UNIQUE ANTIHISTAMINE . . . phenindamine tartrate. Rarely produces drowsiness. Controls the edema and discharge caused by offending allergens.

(3) VITAMIN C. Under body stress such as fever and infectious diseases—the normal requirements double. The daily dose of Vitamin C in Dristan is *more than twice* the recommended daily allowance.

(4) Approved APC formula . . . aspirin, phenacetin and caffeine.

Professional Supplies on Request:
Ask the detail man from Whitehall Laboratories or send request on your professional letterhead to:

References: Annals of Allergy, Vol. 6. July-August 1948. Study of a New Histamine Antagonist, The Pennsylvania Med. J., Vol. 51. Clinical Study of a New Antihistaminic Drug—Phenindamine. Dristan Clinical Study.

WHITEHALL LABORATORIES
22 EAST 40TH STREET, NEW YORK 16, N. Y.

superior topical corticosteroid

In convenient aerosol spray—for the treatment of dermatoses, particularly in out-of-reach problem areas—a fresh approach to therapy

- the superior anti-inflammatory effect of Kenalog Spray[1-7] provides anti-inflammatory, antiallergic, and antipruritic relief in acute, exudative, weeping lesions, even in extensive, out-of-reach problem areas.
- minimal local irritation and less chance of local contamination.
- metabolic studies show electrolyte disturbance does not occur when Kenalog is applied topically.[1, 4, 5]
- easy to apply, gives broad, even coverage, and permits observation of lesions as they heal.

Indications: Atopic dermatitis, contact dermatitis, eczematous dermatitis, neurodermatitis, seborrheic dermatitis, insect bites, pruritus ani and vulvae, lichen simplex chronicus, exfoliative dermatitis, stasis dermatitis, nummular eczema, sunburn.

Dosage: Apply the spray to the affected areas from a distance of 3 to 6 inches, t.i.d. or q.i.d. A 3-second spray (delivering approximately 0.1 mg. of triamcinolone acetonide) covers an area about the size of the hand. *Cover the eyes when using Kenalog Spray on or near the face.*

Supply: Kenalog Spray in 50 and 150 Gm. containers of 3.3 mg. and 10 mg. triamcinolone acetonide respectively. Also available—Kenalog Cream (0.1%), Kenalog Ointment (0.1%) and Kenalog Lotion (0.1%).

References: 1. Reports to the Squibb Institute for Medical Research. 2. Howell, C. M.: Squibb Clin. Res. Notes *1*:5 (Oct.) 1958. 3. Goodman, J. J.: Squibb Clin. Res. Notes *1*:1 (Oct.) 1958. 4. Smith, J. G., Jr.; Zawisza, R. J., and Blank, H.: Squibb Clin. Res. Notes *1*:6 (Oct.) 1958. 5. Fitzpatrick, T. B.; Crowe, F. W., and Walker, S. A.: Squibb Clin. Res. Notes *1*:3 (Oct.) 1958. 6. Lerner, A. B.: Squibb Clin. Res. Notes *1*:2 (Oct.) 1958. 7. Robinson, R. C. V.: Bull. School of Med., U. Maryland 43:54 (July) 1958.

'KENALOG'® IS A SQUIBB TRADEMARK.

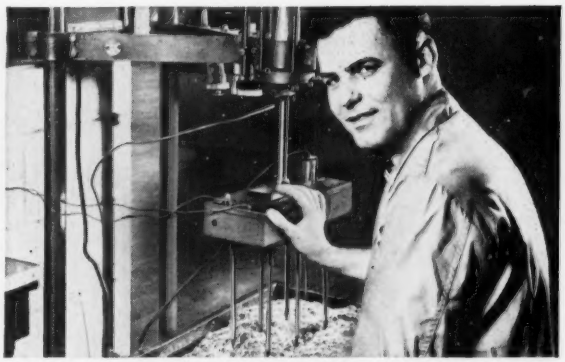
SQUIBB

Squibb Quality—
the Priceless Ingredient

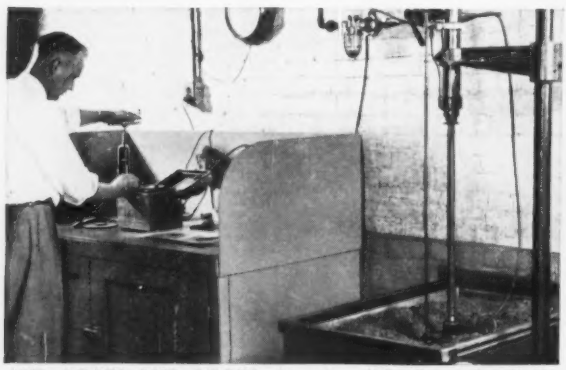
SHOCKING DISCOVERY!

The electrifying secrets of cigarette flavor: 800,000 tests a year to control tobacco moisture

IT'S NOT THE HEAT—IT'S THE HUMIDITY that most affects cigarette taste. The American Tobacco Company developed the "shock test" to control tobacco moisture. Here the prong moisture meter measures electrical conductivity of strip tobacco. Conductivity varies with moisture content.



THE TOBACCO IS TESTED AGAIN by smaller prong moisture meters after shredding. Tests are conducted about every 15 minutes as tobacco goes through the plant. Moisture is not allowed to vary more than a fraction of a per cent (not enough to affect cigarette taste).



AND AGAIN AND AGAIN. At other "check stations" the moisture is retested many times throughout the manufacturing process. These constant tests are essential to quality. When tobacco is too dry, it will not smoke smoothly. When it is too wet, it is flat, tasteless.

MEANWHILE, BACK AT THE LAB, further tests of tobacco samples are made under precisely controlled temperature and humidity conditions. Samples are weighed to the closest milligram, placed in a forced-circulation oven, then a cooling cabinet, then reweighed. Weight loss reveals moisture content.

THE TESTING NEVER STOPS. Even after cigarettes have gone to market, samples are recalled, stripped and rechecked for moisture to determine effects of storage and packaging. All in all, 800,000 moisture tests are made every year. Another example of American Tobacco's incomparable quality control.

The American Tobacco Company

FIRST IN CIGARETTE RESEARCH

©A. T. Co.

90% of anxious, agitated and apathetic office patients calmed without drowsiness and with normal drive restored...

on one or two 0.25 mg. tablets b.i.d.:

This is the pattern of performance for

PERMITIL®

Fluphenazine dihydrochloride

■ In 1164 patients with anxiety and anxiety-induced fatigue or depression, PERMITIL, administered in small daily doses of 0.5 mg. to 1 mg., produced significant improvement in 90%.*

■ PERMITIL is virtually free from side effects at recommended dosage levels.

■ Patients become calm without being drowsy and normal drive is restored.

■ Onset of action is rapid; effect is prolonged. ■ PERMITIL does not potentiate barbiturates or non-barbiturate sedatives and can be used with impunity with such agents.

How to Prescribe PERMITIL: The lowest dose of PERMITIL that will produce the desired clinical effect should be used. The recommended dose for most adults is one 0.25 mg. tablet twice a day (taken morning and afternoon). Increase to two 0.25 mg. tablets twice a day if required. Total daily dosage in excess of 1 mg. should be employed only in patients with relatively severe symptoms which are uncontrolled at lower dosage. In such patients, the total daily dose may be increased to a maximum of 2 mg., given in divided amounts. Complete information concerning the use of PERMITIL is available on request.

Supplied: Tablets, 0.25 mg., bottles of 50 and 500.

*Recent compilation of case reports received by the Medical Department, White Laboratories, Inc.

White WHITE LABORATORIES, INC., *Kenilworth, New Jersey*

Each tablet contains:
Provera (medroxyprogesterone acetate) 2.5 mg.
Cardrase (ethoxzolamide) ..... 35 mg.
Levanil (ectylurea) ..... 300 mg.

DOSAGE: 1 tablet 1 or 2 times daily, 5-10 days before the period.

THE UPJOHN COMPANY / KALAMAZOO, MICHIGAN

CYTRAN*

GETS AT THE CAUSE

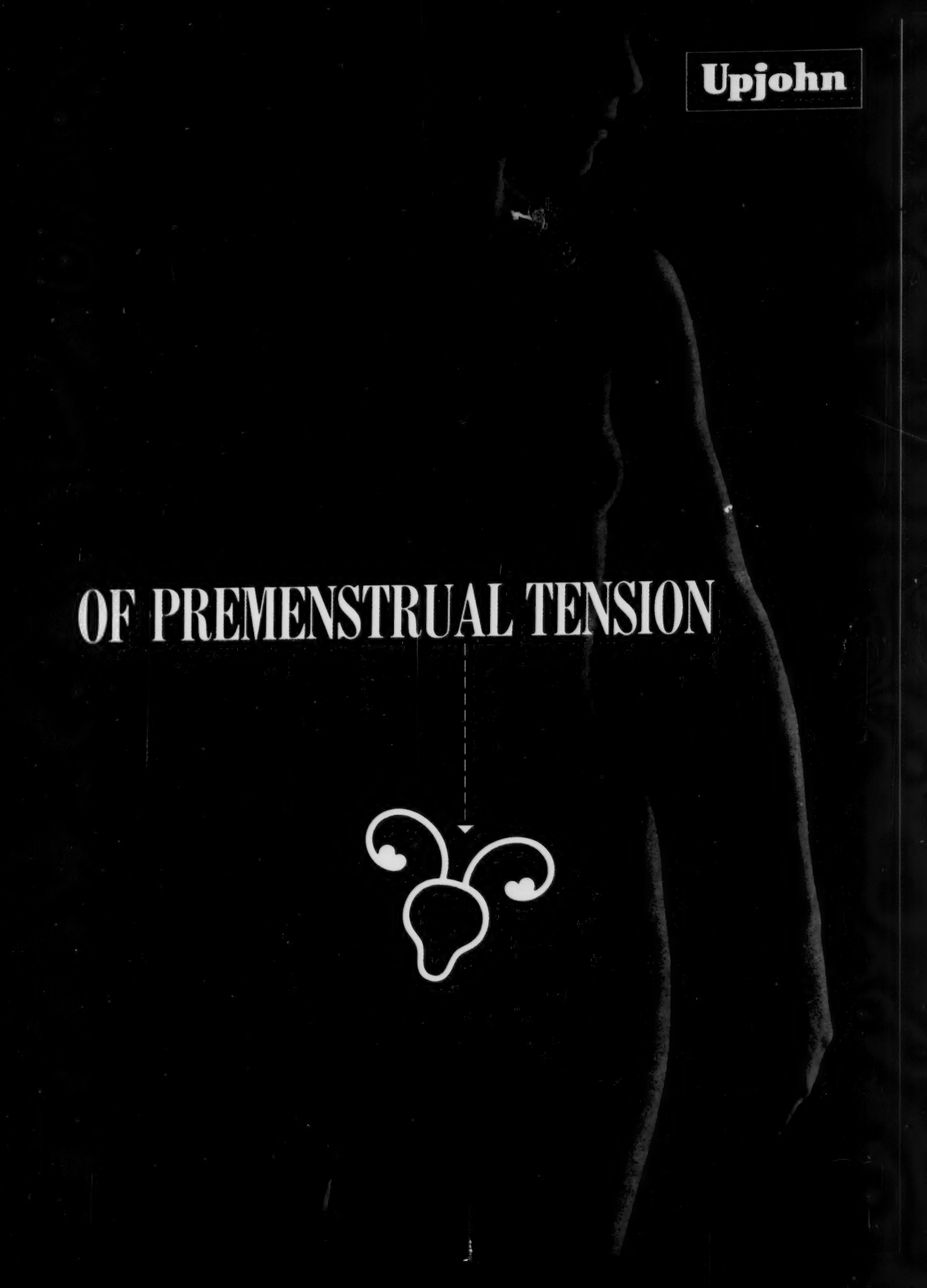
to restore hormonal balance...

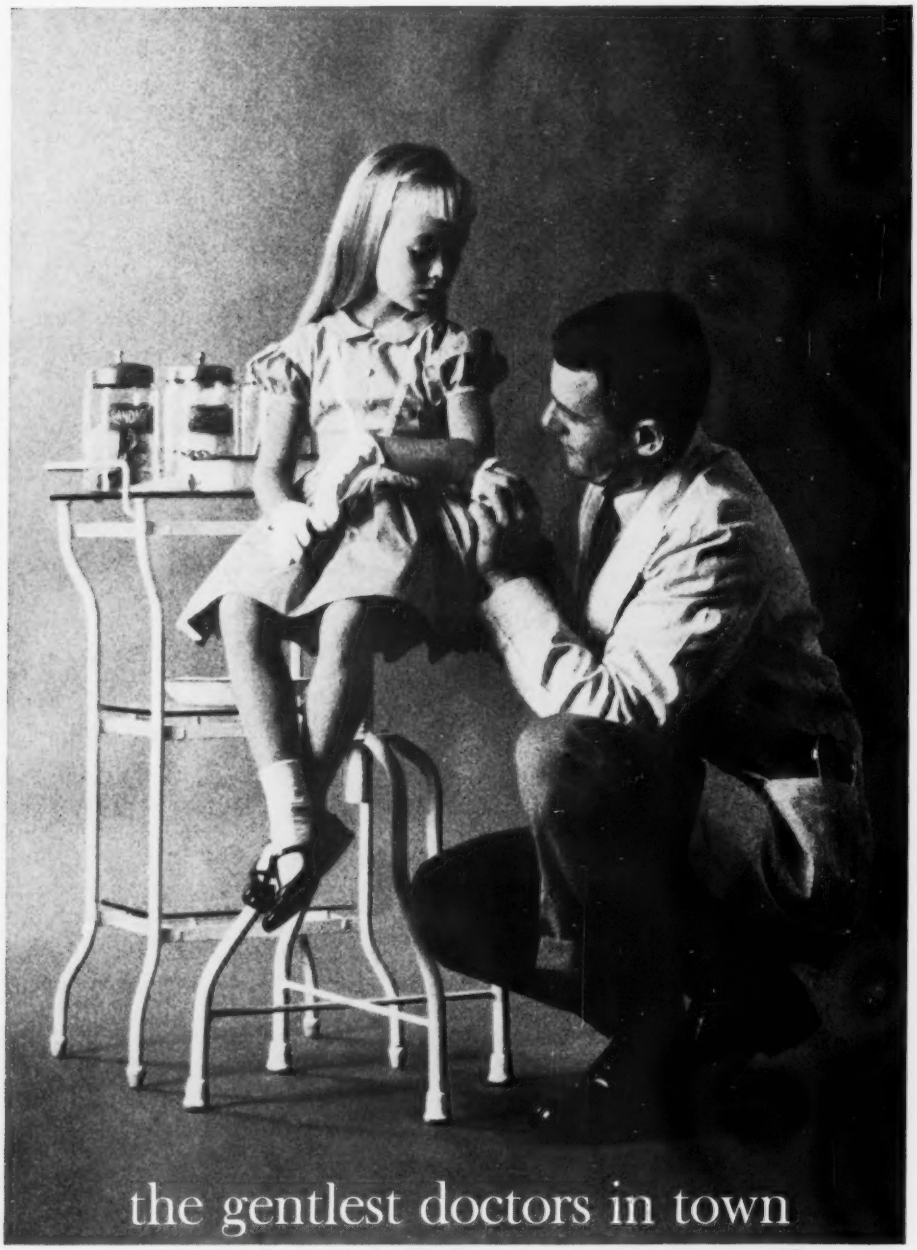
corrective therapy Because Cytran contains the new progestin, Provera,† you can now reach the *cause* of premenstrual tension–hormonal imbalance. Estrogen-progesterone ratio is adjusted to more normal premenstrual balance. Thus even abdominal discomfort, shakiness, fatigue–symptoms incompletely controlled by mere symptomatic treatments–are effectively relieved.

to comfort the patient...

symptomatic therapy An effective diuretic (Cardrase†) and a mild tranquilizer (Levanil†) afford symptomatic relief while Provera works to effect a restoration of hormonal balance. They also supplement the activity of Provera in those rare cases where restoration of hormone balance does not completely eliminate edema and anxiety/tension.

*TRADEMARK †TRADEMARK, REG. U.S. PAT. OFF.


Upjohn
OF PREMENSTRUAL TENSION



the gentlest doctors in town

stop pain with Nupercainal®
(dibucaine CIBA)

...For minor cuts and burns, sunburn, hemorrhoids, removing sutures, performing routine office surgery, making instrument examinations. And, to best suit every situation, there's a choice of Ointment, Cream, Lotion, Suppositories.

CIBA
SUMMIT, N. J.

2/3774MD

Complete information available on request. –

EFFECTIVE THERAPY FOR
TINEA PEDIS (ATHLETE'S FOOT) AND
OTHER RINGWORM INFECTIONS

GRISEOFULVIN®

Griseofulvin

ORAL ANTIFUNGAL AGENT

Before treatment, **T. rubrum** infection.

After 2 months' treatment with GRIFULVIN.

typical response of tinea pedis to GRIFULVIN*

- itching and burning relieved in **2 to 6 days**
- vesicles and scaly patches disappear completely; cultures and KOH scrapings usually become negative in **2 to 6 weeks**
- side effects are rare, mild and transitory

Average dose: 250 mg. q.i.d. Adjunctive treatment with topical keratolytic agents will aid in eradicating the fungi from the skin of the feet.

Supplied: 250 mg. scored tablets, colored aquamarine, imprinted McNEIL, bottles of 16 and 100.

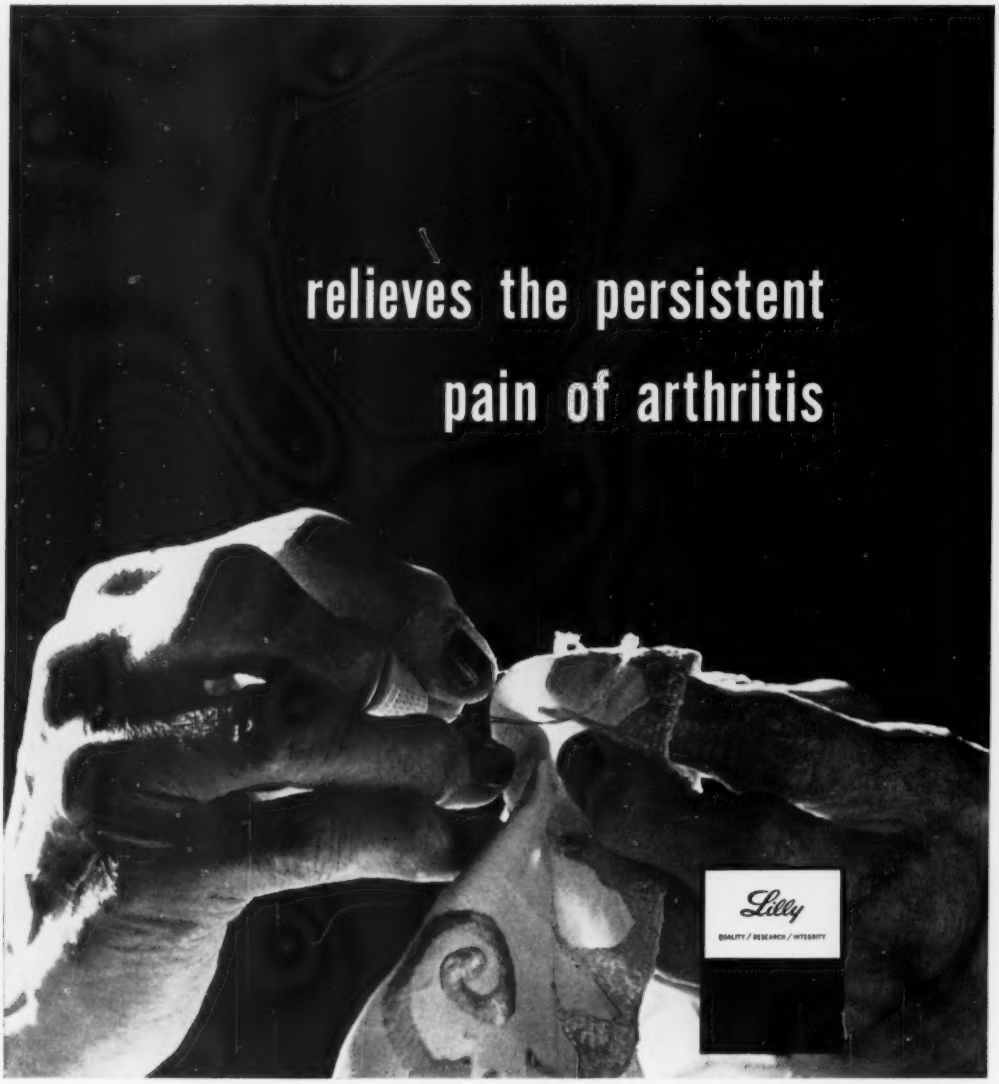
*Blank, H.; Smith, J. G., Jr.; Roth, F. J., Jr., and Zaias, N.: J.A.M.A. **171**:2168 (Dec. 19) 1959.

Photographs courtesy of Harvey Blank, M.D., Miami, Florida

McNEIL

McNEIL LABORATORIES, INC · PHILADELPHIA 32, PA.

324A60



DARVON® COMPOUND

(dextro propoxyphene and acetylsalicylic acid compound, Lilly)

Darvon Compound combines the analgesic action of Darvon® with the anti-inflammatory and antipyretic benefits of A.S.A.® Compound. When inflammation is present, Darvon Compound reduces discomfort to a greater extent than does either analgesic given alone.

Usual dosage: 1 or 2 Pulvules® three or four times daily.
Also available: Darvon, in Pulvules of 32 and 65 mg.

Darvon® (dextro propoxyphene hydrochloride, Lilly)
A.S.A.® Compound (acetylsalicylic acid and acetophenetidin compound, Lilly)

ELI LILLY AND COMPANY • INDIANAPOLIS 6, INDIANA, U.S.A.

020232

IMS

The McIntyre Research Foundation is a nonprofit organization dedicated to the prevention of silicosis and other industrial pulmonary diseases. The Foundation has carried on or sponsored research in the fields of dust control, medical control, and the use of aluminum powder in the over-all program of the eradication of silicosis.

The McIntyre Research Foundation has held 10 conferences during the last 11 years and has provided a common meeting ground on which the Conference participants and the speakers could discuss their mutual problems regarding industrial pulmonary diseases. The researches of the Foundation and of others have been presented at these meetings which have achieved an enviable reputation because of their broad scope. The papers presented at this Conference have helped clarify some of the confusing issues presented by the preventive and diagnostic aspects of industrial lung diseases. Several papers deal with silicosis, one of the most common and controversial of lung diseases. Much of the confusion is caused by the extreme variations in exposures and by the fact that silicosis, once considered a medical disease of the lung, has now become a legal disease and is so defined by the statutes of federal, state, and provincial governments.

Because of the outstanding quality of the papers presented at the 1960 Conference it was agreed to have all papers published by *Industrial Medicine and Surgery*. In this manner the McIntyre Research Foundation could share their new scientific information with the many readers of this Journal.

—J. W. G. HANNON, M.D.
Conference Chairman

INTRODUCTORY ADDRESS

• W. B. DIX
Vice President and Treasurer
McIntyre Porcupine Mines, Limited
Toronto, Ontario

At the conferences of the McIntyre Research Foundation on Silicosis you will usually find many who are directly interested in health conditions associated with the Canadian mining industry. It was in the mining industry in the Province of Ontario, some 25 years ago, that the work of this Foundation was given birth. The original founders had as their objective the mitigation of silicosis as an industrial disease, but the achievement of this goal has involved more time and painstaking effort than they probably ever contemplated. As any student of medicine or science will appreciate, it takes a great deal of intensive study over a period of many years to acquire familiarity with even one subject. To attain and maintain the status of an authority on silicosis is a great challenge. To some extent we hope our Foundation has added something to the knowledge of this disease and the methods of its prevention.

Every industry has its undesirable products. In some they are contaminants and in others they

are by-products of manufacturing processes. No matter what the source, these undesirable by-products must be eliminated and industry spends vast sums of money to do so. In the mining industry, free silica is an undesirable product when found in the atmosphere the miners breathe and must be eradicated or at least reduced to a safe level. The same care which must be given by the manufacturer to eliminate undesirable products must continuously be given by the mining industry to eliminate siliceous-bearing dust. The mining industry has always recognized that silicosis is costly, and it has become more costly as compensable labor has increased and as those receiving compensation are, for the most part, now enjoying a normal life span. The industry's prime concern with silicosis is its effect on the health of its employees. Consequently, mine management has been a great follower of these conferences and I feel sure the knowledge gained has been put to effective use.

For us in Ontario silicosis is still a problem, even though the number of new cases each year has decreased. The trend is encouraging but constant vigilance is the price for success. With this in mind the Minister of Mines for the Province of Ontario in 1957 appointed Dr. John F. Paterson, an independent medical authority and one familiar with chest diseases, to review and report on all matters relating to silicosis in Ontario. His report has just been published and copies may be obtained from the Ontario Department of Mines or the Queen's Printer for the Province. It contains a most excellent review of the situation as it exists today and deals with many aspects of diagnosis, prevention and treatment of the silicotic and tuberculosilicotic.

I should perhaps make some reference here to Dr. Paterson's comments on the use of McIntyre aluminum powder in the prevention of silicosis. Dr. Paterson reports on the difficulty encountered in proving its efficacy in miners. Its value as a preventive in experimental animals has now been proved beyond reasonable question, but to prove to the satisfaction of the medical profession by the use of controlled experiments in miners, with all the variables which would be encountered and the long delay before silicosis might develop, would, I am sure, present so many obstacles as to prohibit a practical demonstration in the Ontario mining industry. It was encouraging to note Dr. Paterson's findings that, according to the Workmen's Compensation Board's records, no miner who had mined in Ontario and who had taken aluminum powder from the time he started mining had developed radiological silicosis up to the end of 1956. Aluminum powder has been used generally in this industry in Ontario for nearly 16 years and while it may be still too early to state that silicosis will not develop because of preventive measures, including dust control, medical control and aluminum powder, the finding is encouraging and we hope it will continue.

The constant care of each and every one of us must include the search for continued knowledge in these subjects. Youth is the time for adventure of the body but more mature age for triumphs of the mind. No greater triumph can be reached than the grasp of something so vast, so complex, and so profound as that which the field of medicine presents. Too often we fail to recognize as classical, a work which passes before us, and all too frequently our common attention to everyday events may disguise a great learning which may become a masterpiece of the future.

Too little knowledge is dangerous, but as long as the willingness to learn remains there is no measuring the heights to which we may ascend. In wisdom and knowledge we can conquer, and today's problems of medicine and science will surely give birth to the glorious happenings of the future. No great plan ever succeeds without meeting and overcoming endless obstacles which are merely sent to try and test our skills and the quality of our courage.

A Practical Method of Providing a

Suitable In-Plant Environment

• K. E. ROBINSON
Ventilation Engineer
Industrial Hygiene Department
General Motors Personnel Staff
Detroit, Michigan

There is considerable interest throughout industry in improving the in-plant environment for the benefit of the employees. This being the case, the problem must necessarily revolve around the employee and his reaction to his environment.

Man is a warm blooded animal with a blood temperature exceeding 98° F. He is most comfortable when lightly dressed in a space maintained at approximately 72°F. providing the air motion is less than 35 feet per minute. When the air temperature varies more than a few degrees either side of 72°F., he becomes uncomfortably warm or cold. If the air motion is increased over his body, he will become chilled unless the air temperature is increased proportionately.

It therefore becomes obvious that the engineer's responsibility is one of controlling the man's rate of heat loss; not, as is often stated, of heating him. When this responsibility is accepted by the engineer, it is possible to develop more practical methods of using air as a medium to improve in-plant environment.

Comfort is usually considered to be attained when the heat gain or loss of the body is so regulated that one is not conscious of heat or cold. About 1912 a physician was invited to talk to the American Society of Heating and Ventilating Engineers. He stated that research should be initiated to find the proper air temperature, air movement, humidity, and radiation for the comfort of man. However, it wasn't until the early twenties that work to determine these factors was actually begun by the Society.

The original studies were carried out by placing seminude people in rooms which were maintained at various air temperatures. Their reactions were recorded and it was determined at what saturated air temperatures most people were comfortable. Consequently, when we refer to a 68° effective temperature, we are speaking of a combination of wet and dry bulb temperatures which has the same effect on the body as 68° saturated air.

Figure 1 shows the comfort chart that resulted from these studies. The effective temperature lines are the curved diagonal lines running toward the lower right section of the chart. These should be of particular interest to the environmental engineer.

The areas on the upper and lower sides of the main chart portray the percentage of people who were comfortable at a given effective temperature. Most favored about 71° for summer and 68° for winter. Again, it must be kept in mind that these figures are for still air only. Although this chart can be challenged, it does serve as a benchmark and will reflect the well-being of the employees.

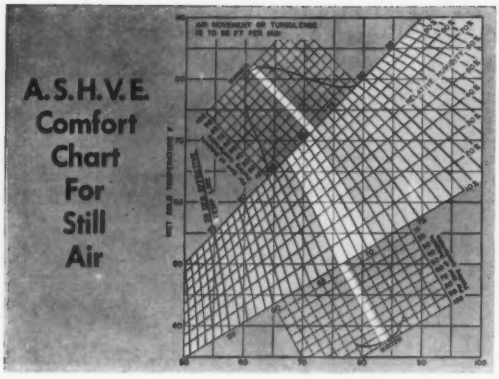


Figure 1

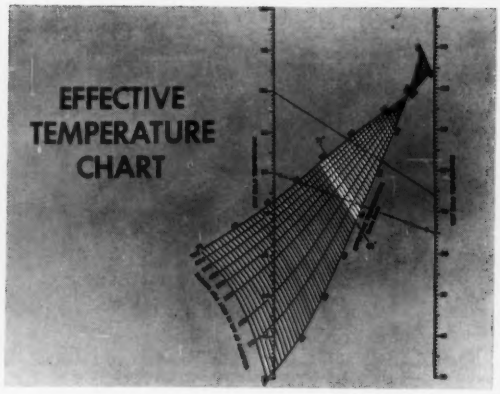


Figure 2

Figure 2 is another form of the effective temperature chart in which air motion has been taken into account. On this chart, velocities from 0 through 700 feet per minute are given. The effective temperature lines for 30 or less feet per minute, which is considered still air, are the same as the effective temperature lines shown on Figure 1. The dotted line represents a condition created by 65° wet bulb and 80° dry bulb. The effective temperature created by this condition varies from 72° to 60° depending upon the velocity to which the man is exposed.

As will be noted, this chart plots the effect of air movement up to 700 feet per minute. The question is often asked, what velocity is best for summer cooling and what is the maximum that can be used. Although it has been stated that there is no additional value to be gained by moving air at more than 800 feet per minute, employees in hot industries are not in complete agreement. Air movement exceeding 3000 feet per minute is often insisted upon. In some plants, small high velocity jets are used so the employees walk in and out of the air pattern. The claim is made that the cooling effect is more noticeable.

Although wet and dry bulb readings, as well as air motion, are generally well understood by personnel, the strain placed on individuals when they are exposed to high energy radiation is often misunderstood. Radiant energy, unlike convected heat, travels in a straight line like light and can be reflected or absorbed. It cannot be controlled by ventilation unless it is first converted to some other form of heat. The construction of an ordinary glass thermometer is such that it reflects the radiant heat and, therefore, does not give a true measurement. The common method of measuring radiant heat is to place a thermometer in the center of a thin-walled black globe. Although 6" copper globes painted a flat black are usually recommended for radiant energy measurements, other sizes will serve satisfactorily for field work. The black globe thermometer, as it is often called, is placed where the temperature reading is desired and left until equilibrium is reached. This will come about when the loss by convection just balances that received by radiation.

Although there are formulas as to how the radiant temperature reading should be utilized in evaluating the working environment, a simple method using the psychrometric chart was developed by Dr. Bedford of England. Later, Professor Hatch of the University of Pittsburgh made further modifications. A psychrometric chart, Figure 3, is necessary, however. To use this chart the wet and dry bulb readings are plotted and at their intersection a line is carried to the right on the equal moisture lines until it intersects with the temperature found by the black globe thermometer.

Example: The readings showed a 65° wet bulb and 80° dry bulb in a plant with a globe thermometer reading of 100°. To determine the synthetic wet bulb reading that we may use on the

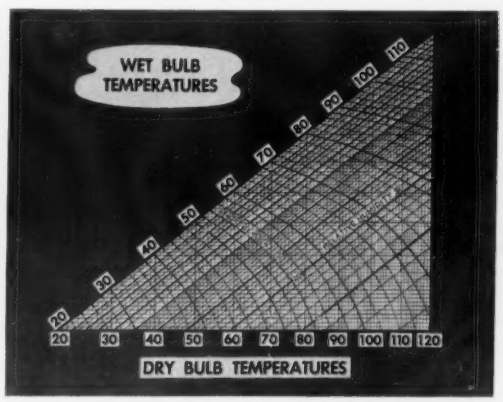


Figure 3

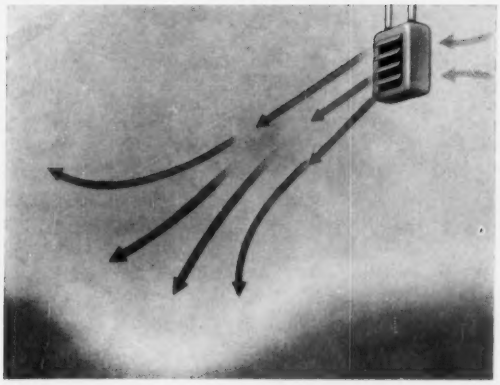


Figure 4

effective temperature chart with the globe thermometer reading, we find the intersection of the 65° wet bulb and 80° dry bulb. The plane is followed upward and to the left and our new wet bulb reading of 71° is determined. By substituting a new wet and dry bulb reading of 71° and 100° in Figure 2, it is noted that the effective temperature in the room will be between 77° and 81° depending on the air movement. Although there has been no standard set as the maximum effective temperature, Dr. Bedford recommends that a maximum of 80° be considered since his research has shown that the rate of production decreases and the rate of accidents and injuries increases when this figure is exceeded for any appreciable length of time.

With this basic understanding of how man reacts to his environment, the engineer is in a position to evaluate different methods of heating and ventilating a plant to obtain a satisfactory environment.

Figure 4 shows one type of heater often used in industry. Attention is called to the deflectors, furnished with this equipment, supposedly to direct the air downward to heat the lower level of the plant. Unfortunately, such a piece of equipment will result in high air motion as well as elevated temperature over any employee stationed on the floor below.

In some areas, particularly the smaller ones such as guard houses, etc., alterations to the unit such as is shown in Figure 5 will eliminate the difficulties and provide a reasonable environment. With this alteration the cold air is drawn off the floor through the ductwork and passed through the unit where it is heated and discharged in a horizontal plane. With such an arrangement, a low pressure area is generated at the floor due to removal of cold air and the warmed air must come down to satisfy this reduction in pressure. This will warm the floor without noticeable air motion on the employee. The ideal arrangement with such a unit would be to modulate the heat input to the heater to satisfy the requirements and to allow the fan to operate continuously, thereby eliminating stratification.

Many industrial operations require additional exhaust to control contaminants generated. Figure 6 shows a cross section of a plant with the typical exhaust fans in the roof as well as a paint booth. The exhausting of large quantities of air results in high infiltration rates at all doors, windows, and other openings. Regardless of how the air from the heating equipment is directed, employees located in the path of the cold air coming into the building, to satisfy the exhaust units, will be chilled and uncomfortable. The addition of extra heaters only results in excessive temperatures in the center of the building but does not control the problem. In many plants one can observe installations of three or four heaters near an outside door and/or canvas, plywood, or glass partitions to prevent infiltrating cold air from reaching the employees. Such makeshift arrangements are temporary and will never be satisfactory.

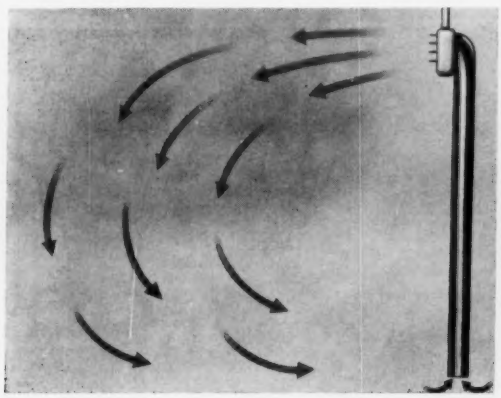


Figure 5

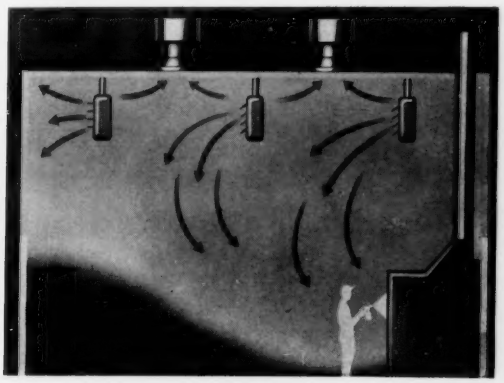


Figure 6

Another way to express the problem is to place it in terms of BTU's or pounds of air that are involved. If a 10-foot square outside door is open for one hour on a zero day it would require approximately 7.5 million BTU's to heat air entering at the rate of 1000 feet per minute. Conditions of this type are not uncommon in plants where the volume of air exhausted from the plant is far in excess of that supplied. This quantity of air would weigh approximately 225 tons and obviously one or more small heaters will have little, if any, effect on this large mass of cold air. The typical recirculating heater located at or above the trusses becomes even less effective than might be anticipated since the fan generates a low pressure area at its inlet which is filled by heated air rather than by the more dense cold air coming through the door.

As more operations are added to the plant, conditions get progressively worse. Figure 7 shows a plan view of the same plant with the paint booth and two heat treat operations which must be ventilated. The furnace gives off considerable quantities of radiant energy which further complicates the problem. The two dots at either end of the furnace represent men who are exposed to high radiant energy on one side and to cold drafts on the other due to the high infiltration rates. The average radiant temperatures might well be 130° or more which will be objectionable to any employee that is stationed close to the furnace. At the same time employees stationed around the exterior of the plant will, undoubtedly, be complaining of excessive cold conditions caused by the high infiltration rate. With such a condition the center of the plant is not only apt to be too warm but also is usually filled with smoke or haze generated by the operations not requiring local ventilation.

Figure 8 shows a method of improving the environmental comfort of employees working during both winter and summer, and at the same time to control the before-mentioned smoke and haze. Fortunately, this method often reduces the total cost of heating as well.

The furnace has been shielded by bright aluminum to reflect the radiant heat back to the furnace rather than exposing nearby employees. Any smoke, fume, or excessive heat that is liberated by the furnace is now retained within the confines of the fire curtain that has been installed where it will be exhausted by the roof fan. The general exhaust fans in the roof of the building have been removed as they are not required in winter. Power fans or gravity installations may be required to exhaust excessive heat in summer depending on type of building and operations carried on.

The most outstanding corrective measure that has been included (Figure 8) is the use of heating units capable of supplying outside air suitably tempered at the 10-foot level of the plant. Air in quantities equal to, or slightly in excess of, that being exhausted is supplied directly to the occupied space through ductwork and special grills. These grills are designed so that the vertical blades can be individually adjusted to direct the air as desired. The horizontal blades are ganged together so they can be quickly adjusted to provide the most satisfactory air flow pattern.

When properly designed, the system is capable of projecting air through the grill at a velocity between 1600 and 2500 feet per minute. The velocity is dependent on length of throw desired and the magnitude of heat generated by the industrial operations. In the winter the grills are adjusted to project the air parallel to the floor or slightly upward if the entering air temperature is below room temperature. In summer one adjustment of the grills will direct the air toward the floor so as to give the employees the cooling effect of high air motion.

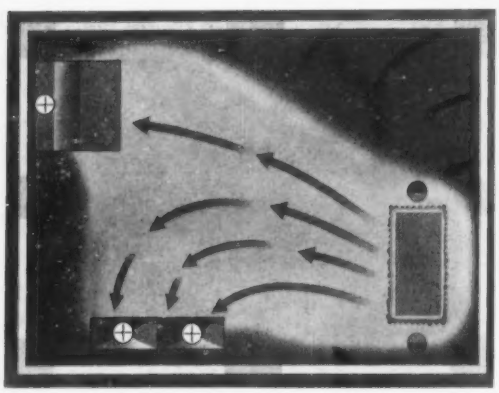


Figure 7

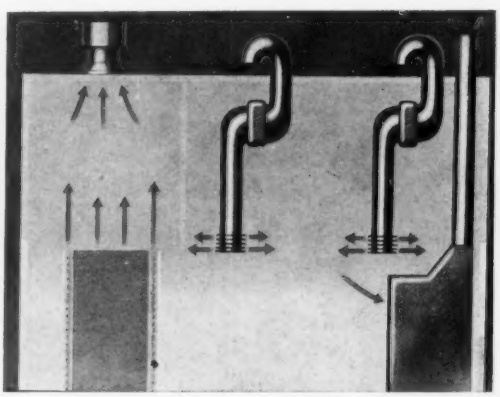


Figure 8

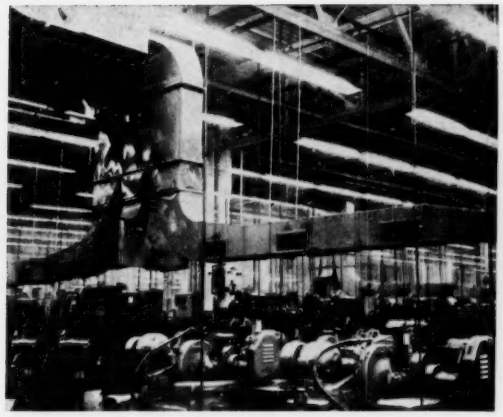


Figure 9

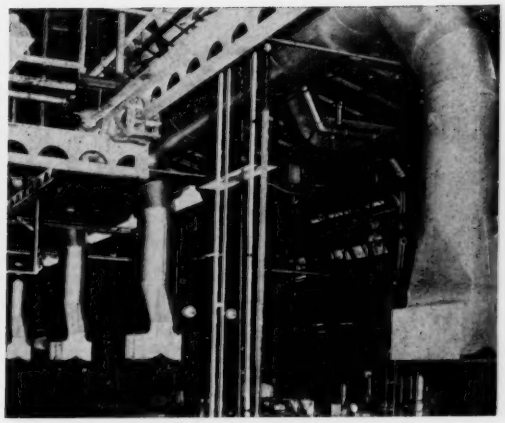


Figure 10

Whereas the winter volumes may vary according to the quantity of air exhausted from the building, the summer volumes usually are calculated at approximately 2 cfm per square foot of floor area. This volume is equivalent to 12 air changes per hour in the lower 10 feet of the plant, this being the level of the registers.

Among the benefits obtained from such an installation is the elimination of high infiltration rates in winter. In addition, reduction of temperatures throughout the plant can be obtained since the air can be supplied sufficiently below room temperature to serve as a cooling media. In the summer the advantages are manyfold. Not only does the employee benefit by high air motion over him, which increases his rate of heat loss, but also the number of air changes in the occupied space for a given volume of air will be considerably greater than if the air is being circulated throughout the entire plant. By delivering the air at the 10-foot level, smoke or heat that are in the upper part of the plant are not re-entrained and discharged on the employees, but rather the contaminants are flushed from the plant through either the gravity or power ventilators through which the supplied air leaves the plant. Whenever the outside temperature declines, as in the evening, the temperature in the plant is correspondingly reduced, thereby eliminating excessive temperatures usually found on second shift operations.

Figures 9 and 10 illustrate methods of distribution that have been used successfully. Figure 11 is the type of grill that has proven most satisfactory. There are no dampers on these grills to reduce the air volume. Double deflecting grills allow good control of the distribution pattern, thereby controlling the quantity and velocity of air that is directed over the employee according to the conditions desired.

In summary, an attempt has been made to describe why many heating systems fail to provide satisfactory environmental conditions in an industrial plant. Methods of providing improved environment throughout the year with the same equipment have been discussed. Designs of satisfactory installations must be based upon the reaction of the individual to his environment rather than the supplying of heat based on the calculated BTU loss from the building. It is believed that installations such as have been described can and will be further refined, and this will result in still further improvement of the in-plant environmental conditions. This will only be possible if the engineer will further consider the reaction of the man to his environment when designing and installing such equipment.

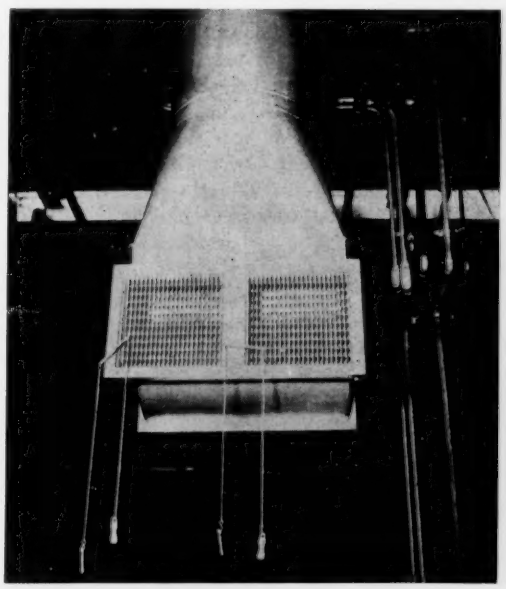


Figure 11

The Clinical Significance of Cavitation in Anthracosilicosis

• PETER A. THEODOS, M.D., F.C.C.P.
Assistant Professor of Clinical Medicine
Jefferson Medical College Hospital
Philadelphia, Pennsylvania

Anthracosilicosis is the type of silicosis found in hard coal miners. It is characterized by a nodular, coalescent nodular or conglomerate form on the x-ray. The conglomerate form is the most common and is manifested by fibrotic masses of varying sizes, generally symmetrically distributed in both lungs. When the distribution is asymmetrical or when localization in one part of the lung occurs, the presence of associated infection is generally considered to be the cause. This infection is frequently tuberculous in nature and it is often accompanied by cavitation. The presence of tuberculosis can be confirmed by recovery of tubercle bacilli from the sputum.

When the tubercle bacillus cannot be recovered from the sputum in the presence of cavitation, then other causes for the cavitation must be sought for. Such things as neoplasm, lung abscess and fungus disease must be considered and if these can be ruled out, then it is most likely that the cavitation is related to ischemic or aseptic necrosis.

Over the past 12 years, 1,847 hard coal miners from the anthracite regions of Pennsylvania have been observed at the Barton Memorial Division of the Jefferson Medical College Hospital. These miners were admitted primarily for evaluation of the nature and degree of the cardiorespiratory impairment resulting from anthracosilicosis. At the same time the presence of cavitation was noted and clinical studies done to evaluate its cause and significance. These studies consisted of a detailed history, physical examination, x-rays of the chest including planigrams and bronchograms where indicated, tuberculin, histoplasm, and coccidioidin skin tests, sputum studies, other selected laboratory studies, and bronchoscopy when required with examination of the bronchial secretions for acid-fast bacilli, bacteriology, fungi and neoplastic cells.

In the history, care was taken to inquire about recent deterioration of the general well-being, the sudden onset of cough with copious black expectoration, fever, chest pain and hemoptysis. Sputum studies for acid-fast bacilli were especially intensive. In the early days of the study the following routine was followed: the initial specimen was smeared and cultured; if negative on smear, then two more plain smears, one 24 hour concentrate, one 48 hour concentrate, and three examinations of the fasting gastric contents with culture of one specimen, were done. Bronchial secretions when obtained were routinely cultured. In the past three years this routine was modified to include only smear and culture of two 24 hour concentrates, as the yield from the large number of specimens previously examined did not justify the intensive study. However, where cavitation was present, the original intensive study of the sputum was carried out if the initial specimen revealed no acid-fast bacilli. In about 85% of cases of anthracosilicosis complicated by tuberculosis as seen in this series, the initial plain smear was positive for acid-fast bacilli.[1]

Since the patients generally were hospitalized for periods of about two weeks, very few died in the hospital. Autopsy study was possible in

only three cases of those considered to have ischemic necrosis. Where done, careful attention was directed towards the area of cavitation with search of the cavity wall and its contents for evidence of tuberculosis.

Definite cavities were seen in the x-rays of 191 cases or 10.3% of the total studied (Table I). Of these, 180 were found on initial admission and 11 on subsequent admission, indicating that they had developed or become manifest during the interim.

TABLE I.
SILICOTIC MINERS WITH CAVITATION

Total cases studied	1847
Cavities on Initial Study	180
Cavities on Subsequent Admission	11
Total cases with cavities	191 (10.3%)

In the 191 cases with cavitation, acid-fast bacilli were found in the sputum by smear or culture in 139, or 72.8%, and the cavitation was considered to be due to tuberculosis (Table II).

TABLE II.
CAVITATION IN SILICOTIC MINERS BY CAUSE

	Number	%
Tuberculosis	139	72.2
Ischemic Necrosis	44	23.0
Bronchogenic Carcinoma	2	1.0
Cystic Bronchiectasis	2	1.0
Undetermined	4	2.2
Total	191	100.0

In the remaining 52 cases, carcinoma of the lung accounted for three cases, cystic bronchiectasis for two cases and ischemic or aseptic necrosis for 44 cases or 23% of the total with cavities. In the remaining four cases the cause could not be determined by the clinical methods used.

In the 139 patients with proved tuberculosis, the majority of cavities were unilateral and single, with about one half displaying a shifting fluid level. Cavitation was multiple in one lung in about 18% and bilateral in about 20%.

In the 44 cases considered to have ischemic necrosis, cavitation was unilateral in 32 cases, and bilateral in 12 cases. In 23 of the 44 cases cavitation was multiple either unilaterally or bilaterally with several cases showing multiple cavities in one lung and single cavities in the other. In one case the presence of cavitation was detected only at autopsy. In 26 patients where serial x-rays were possible over a period of days to years apart, change in size of the cavitation was noted in five cases, with filling in of the cavity and apparent disappearance in seven cases and emptying of the cavity either partially or completely in six additional cases. In eight of this group no change in the cavity occurred under observation, the longest period being 11 years.

The tuberculin test was positive in all but

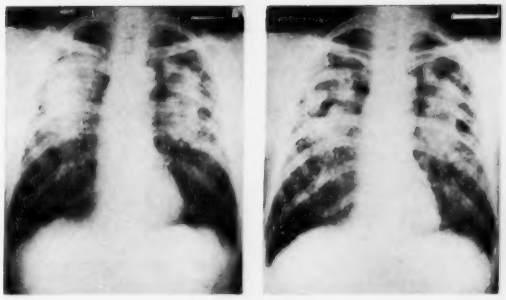


Figure 1

four of the 44 cases with cavitation due to ischemic necrosis. Two of the three classed as negative reactors failed to react to first and intermediate strengths PPD and a third was negative to the first strength. The fourth died before skin testing could be done. In the cases with a positive reaction, 45% were positive to the first strength, 15% to the intermediate strength and 40% to the second strength.

Figure 1 shows the x-rays of a 65 year old miner with typical bilateral conglomerate anthracosilicosis. The film on the left is on admission to the hospital and the one on the right four months later showing the development of a cavity in the right side mass. All sputa were negative by smear and culture for acid-fast bacilli. He has had 11 readmissions over the past 12 years since then with very little change in his general condition and persistence of a negative sputum. Recent films show that the cavity has disappeared on the right side with apparent shrinkage of the conglomerate mass.

Figure 2 is that of a 53 year old miner with massive conglomerate anthracosilicosis showing a fluid level in the right upper mass (left). Five days later he coughed up a large quantity of black material and the film on the right shows complete emptying of the area. All sputa have remained negative and he has remained well.

Figure 3 is that of a 56 year old miner. The x-ray on the left shows advanced conglomerate anthracosilicosis with an air-fluid level at the

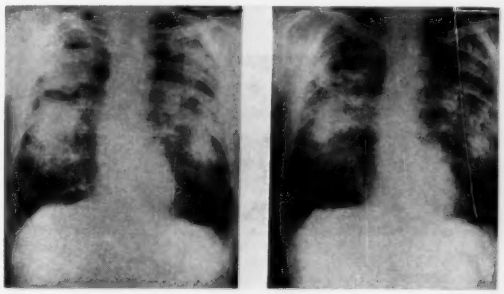


Figure 2

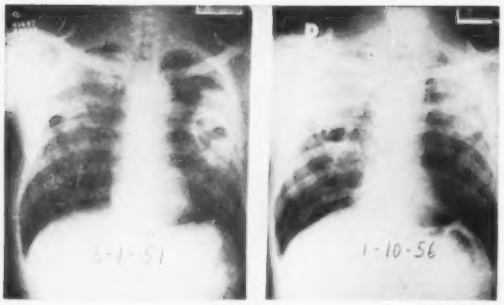


Figure 3

top of the right mass and a moderate sized cavity on the left. The sputum was negative for acid-fast bacilli. He was readmitted four and one-half years later with a nine day history of marked cough and expectoration of a large amount of black sputum. The x-ray on the right shows disappearance of the previously visualized cavities and a new cavity with a fluid level in the lower portion of the right mass. When seen again two years later (Figure 4) the right cavity appeared closed and the left sided cavity was again evident. The sputum has remained negative and he has maintained his state of relative well-being.

At times it is difficult to be sure of cavitation on the conventional film. Planigrams may be helpful by showing a well defined cavity in an area not clearly defined on the plain film.

Bronchograms generally show lack of filling of the excavated areas, implying sealing off of the necrotic area. This is generally a temporary affair as evidenced by the transient nature of fluid levels.

Figure 5 shows the x-ray of a 73 year old hard coal miner for 47 years admitted in a critical condition with chronic blood loss from a bleeding peptic ulcer. No obvious cavitation can be seen on this x-ray (left). He died several days later. At autopsy (right) small cavities containing black inky fluid not communicating with the

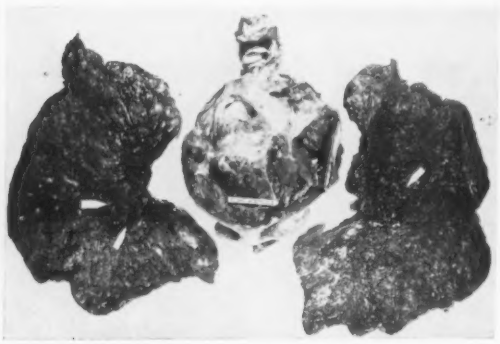


Figure 5

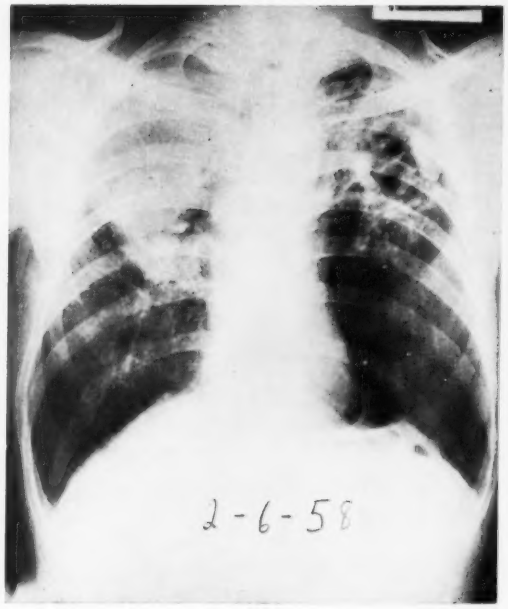


Figure 4

bronchi were found in both lungs. No evidence of tuberculosis was found by culture or histologic study.

Figure 6 shows the x-rays of a 61 year old miner, admitted with cirrhosis of the liver and marked cough and expectoration. The x-ray on the left shows the usual pattern of anthracosilicosis with no evident cavitation. Four weeks later an x-ray (right) showed cavitation in the

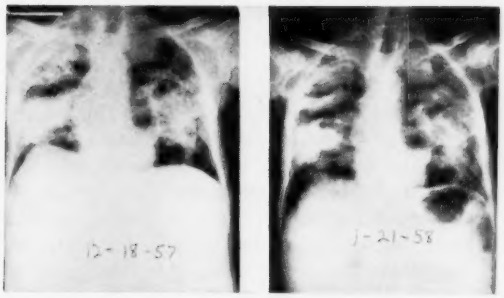


Figure 6

right upper lobe. He died a week later. At autopsy (Figure 7) the right lung revealed three large nodular areas with a cavity measuring seven centimeters in diameter present in the right upper lobe (left). This exuded a thick, black, mucoid fluid into a communicating bronchus. Section of the cavity wall (right) showed no evidence of tuberculosis and smears of the cavity contents revealed no acid-fast bacilli on smear and culture.

In the presence of cavitation, the possibility of tuberculosis must always be considered and diligent and persistent search of the sputum for acid-fast bacilli must be done routinely. In the case of a 48 year old miner an admission x-ray showed advanced conglomerate anthracosilicosis and the sputum was negative. On readmission two and one-half years later breakdown of the left upper conglomerate area was evident. Because of the absence of symptoms, his general well-being and several negative plain smears, the possibility of ischemic necrosis was entertained. However, a sputum culture was reported as positive six weeks later indicating that the cavitation was tuberculous in nature.

In this series of 1,847 cases, the diagnosis of associated tuberculosis was established in 279 cases, or 15.1%, by recovery of acid-fast bacilli by smear or culture in the sputum. Of these, 139, or about 50%, showed cavities and it can be assumed that the cavitation was due to tuberculosis.

When cavitation without a positive sputum is present it would appear likely that the cause is ischemic or aseptic necrosis. Numerous reports have called attention to the presence of aseptic cavitation in the lungs of coal miners who have come to autopsy.[2,3,4,5] This type of cavitation may result from a number of factors, of which the most important appears to be vascular. Investigators have demonstrated that the blood vessels in the massive conglomerate areas are frequently obliterated by an endarteritis obliterans with thickening of the intima and narrowing of the lumen.[6] The resulting anemia in the depth of the lesion is followed by areas of local necrosis which because of autolysis ultimately liquefy and excavate. This may replace part or all of the involved area and only become evident when communication with a bronchus becomes established and emptying of the liquid contents occurs. The resulting cavity may stay patent for many years, may contract and eventually disappear, or may be filled and appear as a solid mass. At other times it may be completely empty.

The pathologic finding in ischemic necrosis in cavitation of varying sizes filled with inky black material from which tubercle bacilli and other organisms cannot be recovered. The cavity wall and surrounding tissue show no evidence of cessation necrosis, round cell infiltration or giant cells.

Figure 7

The usual clinical course of ischemic necrosis is one of a relatively benign sequence of events without evidence of toxicity or effect on the general disability. The development of the necrotic area is generally unsuspected until emptying occurs. This is evidenced by a sudden spontaneous expectoration of large quantities of black sputum (melanoptysis) which is frequently described as up to "a bucket a day."

It is possible that many so-called conglomerate areas are actually filled in necrotic areas. The French have utilized transthoracic puncture to show in some cases that the conglomerate shadows on x-ray correspond to encysted intrapulmonary collections of fluid.[7]

The nature of the cavitation alters the prognosis. Of the 139 patients with tuberculous cavitation, 56 or 40% have died, the great majority (80%) within three years of the diagnosis. Seventeen of the 44 cases of ischemic necrosis (39%) have died from periods ranging from several days to eight years after the initial diagnosis of cavitation, 65% within the first three years. However, since cavitation was present in all but one of these 17 cases at times of initial admission to the hospital it is impossible to say how long the cavitation was present before death occurred. Death in these cases was generally related to the degree of fibrosis, the effects of the associated emphysema and cardiac failure, particularly secondary to cor pulmonale. Death occurs in relation to the severity of these factors and not to the size or number of the cavities.

Treatment of silicosis with tuberculosis is similar to that for tuberculosis in general. Chemotherapy has been effective in controlling the manifestations of toxemia but has had very little effect on cavity closure and conversion of sputum. Collapse measures have seldom produced cavity closure and their use is limited by the reduced pulmonary reserve. No specific treatment is required when cavitation due to ischemic necrosis is present. Treatment is similar to the non cavitated cases utilizing such modalities as bronchodilators, cardiac drugs, antibiotics and intermittent positive pressure breathing.

Recognition of the nontuberculous nature of cavitation is important so that unnecessary hospitalization in a tuberculosis facility can be avoided and the use of IPPB not withheld.

A miner with ischemic necrosis can be permitted to live in normal relationship to his family and community and may even be gainfully employed depending on the adequacy of his pulmonary function.

References

1. Theodos, P. A., and Gordon, B.: Tuberculosis in Anthracosilicosis. *Am. Rev. of Tuberc.*, 65:1, 1952.
2. Schwartz, S., and Auerbach, O.: Non-Tuberculous Pulmonary Disease Simulating Tuberculosis. *Quart. Bul.* Sea View Hospital, Vol. 4, No. 1, 1938.
3. Slesinger, H. A.: Anthracosilicosis in Bituminous Coal Miners. *Arch. Indust. Hygiene and Occup. Med.*, 2:284, 1950.
4. Gough, J.: Pneumoconiosis in Coal Trimmers. *J. Path. and Bact.*, 51:277, 1940.
5. Wall, N. M.: Anthracosilicosis with Special Reference to Pulmonary Cavitation. *Am. Rev. of Tub. and Pulm. Dis.*, 71:4, April 1955.
6. Vorwald, A. T.: Cavities in the Silicotic Lung: A Pathological Study with Clinical Correlation. *Am. J. Path.* 17:709, 1941.
7. Gerneq-Rieux, C., Balgairies, E., Fournier, P., and Voisin, C.: Semaine Hop. Paris 34:1081, April, 1958.

Worth a Pound of Cure

The man who led the Salk polio vaccine field trials urges the nation's physicians to add a new perspective to their traditional practice of preventive medicine. Dr. Thomas Francis Jr., Chairman of the Department of Epidemiology at the University of Michigan, recommends an attack upon the chronic diseases — arthritis, rheumatism, heart disease and others — using the principles of preventive medicine that have proved effective against contagious diseases: (1) *Protection* from chemical and physical exposure; (2) *Correction* of dietary deficiency or excess; and (3) *Sanitation, immunization* and other specific counter-measures to prevent infectious disease.

In an article in the *Journal of the American Medical Association*, Dr. Francis chides the health and medical professions for "the commonly repeated assurances that infectious disease is conquered." He says this "is simply not in keeping with the evidence."

The U of M physician holds that medicine must maintain efforts to prevent contagious diseases and environmental hazards, but at the same time must broaden its research goals toward ultimate "prevention" of the chronic and wasting diseases.

He says, "Preventive medicine will be the medicine of the future, only if it accepts its proper responsibility. Research must be increased in the area of the common annoying disorders of health in which preventive promise is high."

Diffuse Lung Lesions That Mimic Pneumoconiosis

• HOWARD S. VAN ORDSTRAND, M.D., F.C.C.P.
Cleveland Clinic Foundation
Cleveland, Ohio

Man is often prone to blame the environment for his illnesses, particularly his respiratory troubles. Usually it is not a willful blame, but a subconscious mechanism. My colleagues in psychiatry well understand this; they call it "ego projection." The boundary between occupationally caused disease and nonoccupationally caused disease must be carefully defined, and I am sure that I speak for all of us in attendance at this Conference when I say that we want to see justice done. The worker who has an occupational disease should be properly diagnosed, treated, and compensated.

However, to complicate matters, we are more and more recognizing, especially in recent years, that new, or relatively new, nonoccupational diffuse lung diseases have been discovered, or have become better recognized, just as new types of pneumoconiosis have been reported. Some of these new diseases are symptomatic and some are not. The frequent use of chest x-ray studies and mobile-unit surveys, as part of a general examination, have led to the recognition of a number of such nonoccupational diseases. Improved diagnostic methods, including surgical lung biopsy techniques, have clarified hitherto questionable etiologies. Newly recognized diffuse lung diseases, of recent years, which appear to have no environmental causation, include the Hamman-Rich syndrome, pulmonary alveolar proteinosis, and pulmonary alveolar microlithiasis. These may in some instances strongly mimic dust disease of the lungs, just as do some of the better known diffuse entities such as certain lesions of scleroderma, polyarteritis, lymphoma, sarcoidosis, bronchiolar carcinoma, and "miliary" metastatic malignant neoplasms.

The above-mentioned nonoccupational problems occur in all walks of life and, in certain instances, can mimic the roentgen evidence of pneumoconiosis or, if occurring in workers in dusty trades, may present the additional problem of a possible job relationship. Not all of the members of the medical profession have had ample training to appreciate the diagnostic requirements relative to pneumoconiosis; namely, (1) roentgen evidence typical of such lesions, and (2) an adequate occupational exposure. Therefore, it was believed that it would be of interest to present to you representative examples of some of the diffuse lung lesions that, because of their evidence on roentgenograms, or because both of roentgen appearance and of suspicious occupational association, can to some degree mimic pneumoconiosis.

Representative Diffuse Mimetic Lesions

Figure 1 is a chest roentgenogram of a 50-year-old man showing evidence of a predominantly symmetric infiltration of the lower lung fields, which to some degree mimics asbestosis. This man had worked in the insulation-manufacturing industry for 15 years. Both he and his family physician strongly suspected that his disease was of work relationship. His symptoms were a progressively persistent cough and shortness of breath over a five-year period, during which time he also experienced a decrease in appetite and a 20-pound loss in weight. Studies of tissues

from a surgical lung biopsy showed the typical histologic picture of the nongranulomatous fibrosing interstitial pneumonitis of the Hamman-Rich syndrome. The patient died two years later; the diagnosis was confirmed at necropsy.

Figure 2A shows the roentgen evidence of a bilateral diffuse pulmonary lesion in a 42-year-old man with an occupational history in a dusty trade. Although the lesion is of different roentgen appearance from that in Figure 1, this patient on diagnostic study, which included tissues from a lung biopsy, also was proved to have the Hamman-Rich syndrome, as shown histologically in Figure 2B. This patient has improved clinically on prolonged steroid therapy.

Figure 3A is a chest roentgenogram of a 34-year-old woman who was first examined because for the previous one and one-half years she had become increasingly short of breath on exertion. She had a slightly productive cough. Her occupational history was essentially negative, other than that 15 years previously she worked for a six-month period at an electric company where her job was making cores for sewing machines. She had known of the roentgen evidence of the diffuse abnormality for more than two years. The diagnostic study, including lung biopsy, showed the microscopic picture of the currently recognized entity of pulmonary alveolar proteinosis (Figure 3B).

Figure 4 is a chest roentgenogram of a 40-year-old soft-coal miner who was examined because of the development of a relatively non-

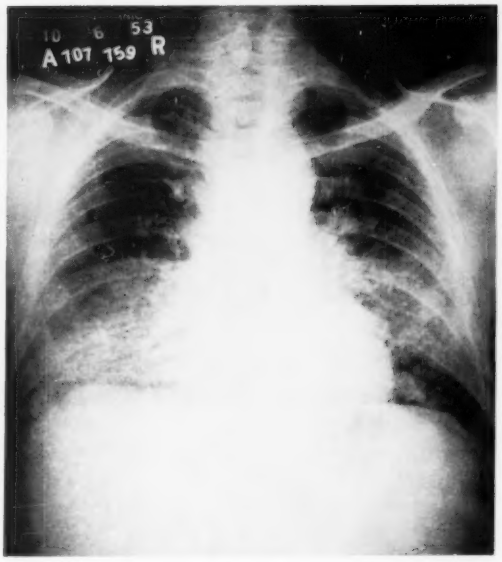
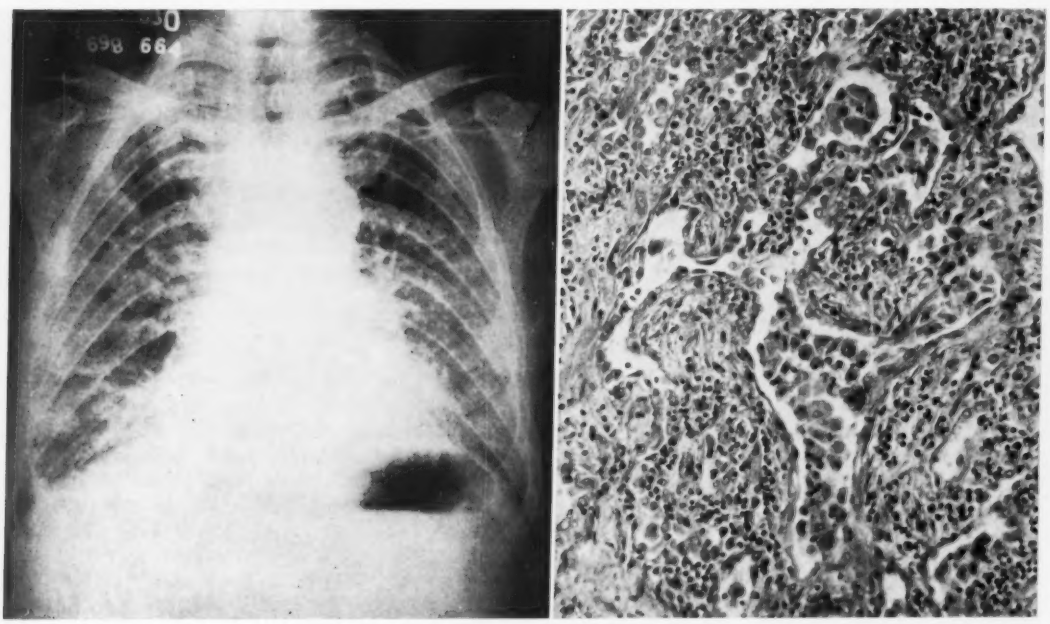


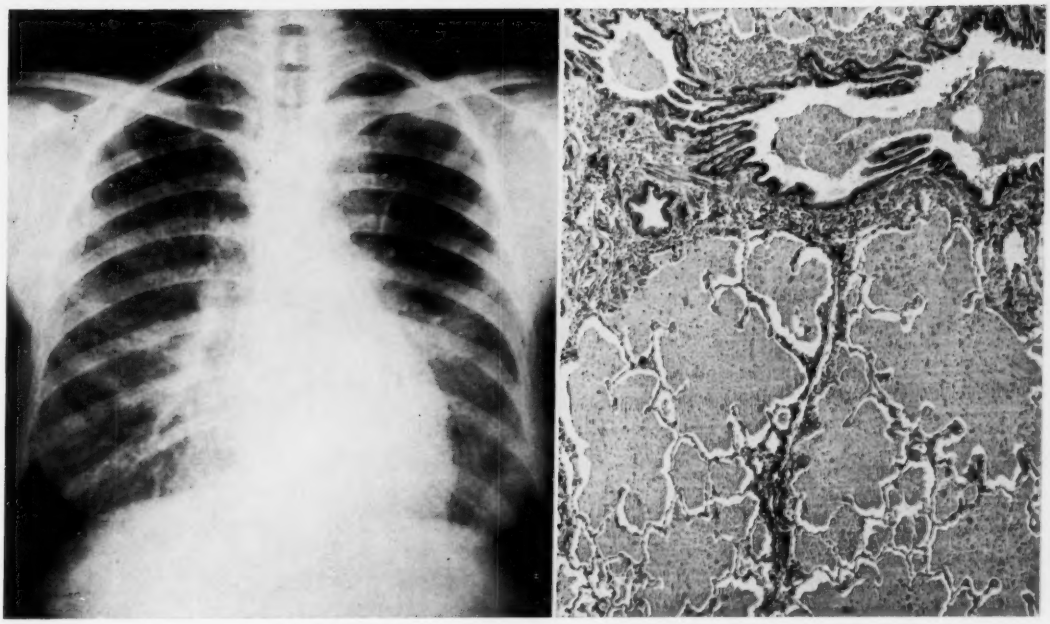
Figure 1

productive cough and progressive shortness of breath during the previous year. From the diagnostic studies the diffuse pulmonary lesion was suspected of being "pulmonary scleroderma," the pulmonary manifestation of visceral sclerosis. The patient died two years later; at postmortem examination this diagnosis was confirmed.

Figure 5 is a chest roentgenogram of a 38-



A Figure 2 B



A Figure 3 B

year-old housewife who was asymptomatic. She had worked for 10 years in a pottery. The diagnostic studies, which included those of specimens of a lung biopsy, showed that the roentgen evidence of a diffuse lesion was due to a parenchymal form of pulmonary sarcoidosis. Subsequently, spontaneous clearing occurred.

Figure 6A is a chest roentgenogram of a 58-year-old man who was relatively asymptomatic when he was first examined. His occupational history was negative, other than that 15 years previously he had been a molder in a foundry for five years. Subsequent to the initial examination, rapidly progressive symptoms developed in the patient, and he died six months later. At necropsy examination the diagnosis of bronchiolar carcinoma was established; the pulmonary histology is shown in Figure 6B.

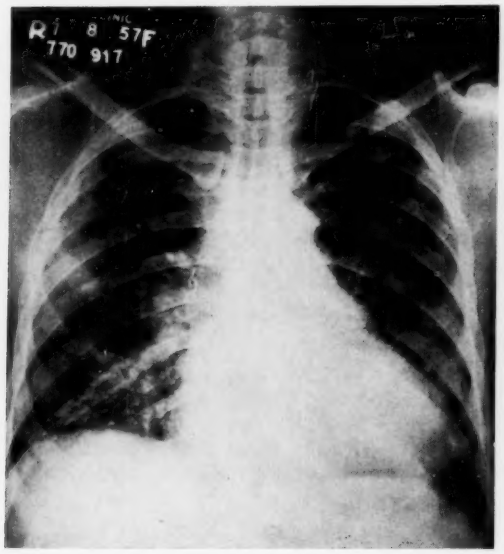


Figure 4

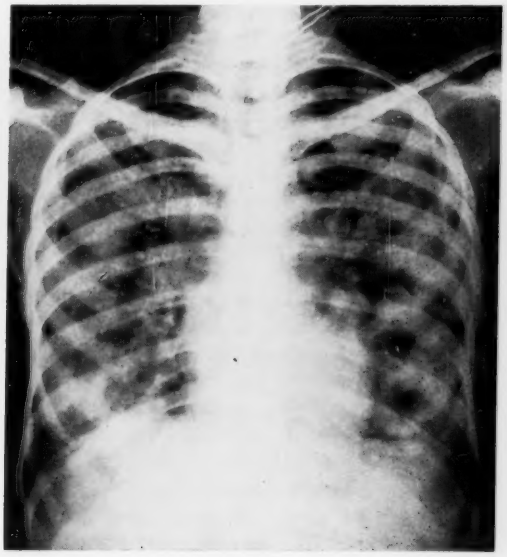


Figure 5

A Figure 6 B

Figure 7 is a chest roentgenogram of a 48-year-old railroad worker who was first examined because of symptoms of mild shortness of breath of one year's duration. The roentgenogram shows evidence of diffuse, and at least partially calcified nodulations throughout the lung field, except in the right upper lobe where the relative asymmetry is due to large bullae. Pulmonary function studies showed the pattern of a mild degree of diffuse obstructive emphysema. This man's entire occupational history was of 28 years as a welder for the railroad. Studies of a specimen of a surgical lung biopsy showed that the nodules were scars of histoplasmosis. No evidence for a pneumoconiosis was found.

Summary

Although new types of pneumoconiosis have been reported in recent years, nonoccupational diffuse lung diseases have also been discovered, or have become better recognized. Some of the latter diseases can occur in workers in dusty trades, and the roentgen appearance of the lesions may mimic the roentgen evidence of occupational diseases of the chest. Some diffuse lung diseases that can so suggest a work relationship include the Hamman-Rich syndrome, pulmonary alveolar proteinosis, pulmonary alveolar microlithiasis, the collagen diseases, lymphomas, sarcoidosis, bronchiolar carcinoma, and metastatic malignant neoplasms. Examples of such mimicry have been shown.

At the conference, roentgen evidence and histologic examples were also shown of diffuse lesions due to mineral oil, periarteritis, miliary carcinomatosis, lymphoma, and farmer's lung, along with illustrations of comparable roentgenograms of some of the occupational lung diseases, including stannic oxide pneumoconiosis, talc pneumoconiosis, asbestosis, and both chronic berylliosis and the acute chemical pneumonitis of beryllium workers.

Figure 7

The Value of
LARGE LUNG SECTIONS
in The Study of
DISEASES OF THE CHEST

• WILLIAM TAYLOR, M.D., C.M., F.C.C.P.
Medical Director
Miners' Chest Examining Station
Timmins, Ontario

In the summer of 1946 it was my good fortune to have Prof. Jethro Gough of the Welsh National School of Medicine visit me at the Miners' Chest Examining Station in Timmins, Ontario, Canada.

During this visit Prof. Gough showed and discussed the diagnostic and educational merits of a series of large lung sections that he had recently processed.

The pathological specimens were saggital sections of the whole lung, about 450-500 Mμ in thickness, unstained and mounted on paper. By virtue of their paper mounting the specimens could be filed with the clinical records, and, if so wished, serial specimens could be filed in ordinary metal filing cases. It is of interest that one drawer of a filing cabinet would hold approximately 1,000 of the mounted sections.

Another advantage of the large sections was that they could be viewed by an ordinary x-ray view box, and one could see the distribution of any pathological changes that may be present. This is especially of interest in diffuse pulmonary disease whether it be fibrotic changes, emphysema, infection, etc.

The study of these large lung sections is particularly interesting and valuable for the following reasons:

1. Their value for teaching purposes at medical schools throughout the country. The diagnosis of silicosis is generally poorly understood, as silicosis is seldom seen except by industrial physicians and those working in mining communities. It is a rare condition of which many physicians know little.

2. The correlation of the diagnosis established during life with the findings at autopsy.

3. Their value in settling disputed claims for compensation purposes.

(a) Claims that were not allowed during the life of the claimant because of negative medical and radiological findings.

(b) Establishing the cause of death in recognized claims. The Workmen's Compensation Board of Ontario requires that the cause of death be directly related to the claimant's compensable condition, before the death claim is allowed.

(c) In cases where there is no dispute regarding the death claim, it is of great value to the doctor to be able to correlate the findings of the case during life and after death. It is only by this method that progress in medicine is possible.

Dr. Gough* in a discussion with the McIntyre Research Foundation in 1952 suggested that as part of its research program on silicosis it might consider facilitating the accumulation of a national collection of representative types of pneumoconioses and other diseases of the

*GOUGH, J. and WENTWORTH, J.: *Jr. Roy. Microscope Soc.* 69:231, (1949)

lung. The directors of the McIntyre Research Foundation readily accepted Dr. Gough's suggestion and offered to grant the necessary funds to carry out this work. The cost to the foundation to date has been $14,000.00. This figure is actually much higher as some of those connected with the project refused financial remuneration.

The technique for preparing the large lung sections was fully explained and demonstrated by Dr. Gough when he addressed the annual McIntyre Research Foundation Conference in Noranda, Quebec, in 1952.

For those interested, the Foundation has made available reprints of Dr. Gough's technique for preparing these large lung sections.

Discussions were then held with officials of the Department of Health of the Province of Ontario, particularly with Dr. Grant Cunningham, Director of the Division of Industrial Hygiene. Dr. A. R. Riddell, Senior Medical Specialist with the Ontario Department of Health, very kindly consented to arrange for the collection of material and directing the work, and Dr. A. J. Blanchard, Chief Pathologist at Sunnybrooke Hospital, prepared the sections (see appendix).

It is of interest that at the penumoconiosis conference held in Johannesburg, South Africa, in February, 1959, two of the many recommendations made were:

1. A clearer demonstration of pathological anatomy can be obtained by cutting Gough and Wentworth sections of expanded lungs. The technique should be used routinely wherever possible, especially when radiological and pulmonary function intercorrelations are to be made with the pathological anatomy.

2. That correlation of radiological and pathological changes should be made on as many and as representative samples as possible, to provide additional information on

(a) early diagnosis of pneumoconiosis

(b) the differentiation of the radiological appearance of pneumoconiosis from those of other conditions.

Paper mounted whole lung sections should be filed with the corresponding radiographs for easy reference and research.

At my office we have continued this type of study for the past few years, and of course the study basically depends on obtaining autopsy material. In Ontario an autopsy can only be performed after written consent of the nearest relative (usually the widow), or in the matter of death by misadventure, an autopsy can be authorized by the Attorney General of the Province of Ontario.

In our area when a claimant dies and there is question of the status of the claim, or if the relatives of the deceased desire to have an autopsy performed, permission is obtained through the family physician. The Workmen's Compensation Board of Ontario has no legal authority to order an autopsy in any case.

In doing an autopsy, the trachea should be sectioned as high as possible. This enables us to suspend the lungs so that the entire bronchial tree can be filled with fluid by gravity (see page 248). In addition to Dr. Gough's technique, I believe it would be of great value if the medical man who is familiar with the clinical and radiological findings of the case is able to section any conglomerate lesions of the fresh lung, for checking the presence of tubercle bacilli. Formalin negates this important part of the investigation.

As I have been stating for years, the presence of complicating pulmonary tuberculosis is the main problem of disability in our silicotics.

After the material has been fixed, and this takes about two weeks, it is sent to Dr. A. R. Riddell* of Toronto, along with the local clinical and radiological information. He, in turn, sends the lungs to Dr. A. J. Blanchard*, who after his pathological examination, provides us with a description of the lungs, both macroscopic and microscopic, a summary of his findings along with the large lung sections.

Incidentally, these sections are not stained; they represent a thin slice of the actual lung as removed from the body.

The large lung sections (Figures 1 and 2) demonstrate carbonaceous material in the lungs and hilar glands as well as the silicotic nodules and emphysematous changes that are present. The photographic cuts cannot be viewed in x-ray view boxes as can the original sections, and thus they lose some of their diagnostic value.

In our studies we have been able to demonstrate examples of bronchopneumonia; bullous emphysema in a nonsilicotic; diffuse vesicular emphysema in a nonsilicotic; diffuse and focal emphysema with chronic bronchitis in a silicotic; lung abscess; lung embolism with infarction; primary carcinoma of the lung; hamartoma of lung; pulmonary tuberculosis; simple silicosis; silicosis with pulmonary tuberculosis; fungus disease of the lung; cystic bronchiolectasis of the lung; examples of granite cutters lung; molders lung, coal miners lung, etc.

It is my opinion that the use of the Gough large lung sections is a valuable contribution to the study of diseases of the chest.

---

*Blanchard, A. J., and Riddell, A. R.: Preliminary experience with the Gough Large Lung Section Technique in the Study of Silicosis and other Pulmonary Dust Conditions. McIntyre Research Conference 1954.

Figure 1

Figure 2

Large Lung Section Technique
Blanchard Modification of the Gough Technique

Remove the lungs from the body whole and without rupturing the pleura. If there are dense adhesions take the parietal pleura out with the lung. Cut off at the hilum and fully distend by running the following solution into the major bronchi by means of a tube and cannula from a reservoir about four feet above the lung.

Liquid Formaldehyde (40%)	500 cc
Sodium Acetate	200 gms
Water	5000 cc

When the lung is fully distended the bronchus is tied off securely so that the fixative does not escape. The lung is placed in a container of the above fixative large enough for it to float freely with no distortion from pressure. Cover with a cloth wet with the fixative. The amount of fixative necessary to distend the lung varies up to about two liters and in the container we use there is a further three to four liters.

Fix for one week or longer and then cut a slice about one inch thick. This cut is made in the most suitable direction for the demonstration desired. It is desirable to have the slice cut at an even thickness throughout its entirety.

Wash the slice in running water for about 72 hours to remove the formaline and place in the following solution:

Gelatin	250 gms
Cellosolve (Ethylene Glycol Monoethyl ether)	20 cc
Caprylic Alcohol	5 cc
Water	850 cc

The slice is placed in the gelatin solution at a temperature of 55° to 60° C. We find a vacuum dessicator, 250 mm in diameter is a satisfactory container for this purpose, because, as a partial vacuum is created, the removal of air from the slice is facilitated and this in turn assists the penetration by the gelatin. Sufficient air can be removed within an hour during which time the gelatin remains fluid at ordinary room temperature. The specimen is then kept in this gelatin solution in an incubator at 37° C for about 72 hours, care being taken that the specimen is completely immersed and the container covered to prevent evaporation.

When impregnation is complete, the specimen is cast in a block by immersing it in melted gelatin in a shallow dish and allowing the gelatin to harden. In casting, it is important that the specimen be kept as flat as possible, especially the surface to be cut. This can be accomplished by weighting the specimen against the bottom of the dish. It is beneficial to allow the gelatin block to set in the refrigerator for about 24 hours. Then the gelatin is firmly set and has shrunk enough that it is readily removed from the dish. The block is trimmed and then fixed to the microtome holder by warming the latter and then putting weights on top of the block until the gelatin sets and sticks the block to the holder. The block fixed to the holder is then placed in a freezer at −15° C overnight or until sections are to be cut.

The microtome used is the large section microtome designed by Gough and Wentworth. It is manufactured and sold by Measuring and Scientific Equipment Ltd., London, England, or may be obtained through their Canadian agents, Messrs. Cave and Company, Vancouver, B.C.

When sections are to be cut the microtome is prepared and pans of water at refrigerator temperature are made available. The block frozen to the microtome holder is removed from the freezer and attached to the microtome. Sections are cut as thawing takes place. A warm towel is rubbed on the surface to hasten thawing. Do not cut sections until the block is sufficiently thawed to cut easily. Sections are cut at about 400 microns but it may be necessary to cut thicker or thinner depending on the nature of the specimen. With a block of tissue one inch thick, up to 40 satisfactory sections may be obtained. When cut, sections are transferred to cold water and placed in refrigerator. Gough recommends placing sections in 10% formalin for 24 to 48 hours to harden gelatin and then washing one to two hours before mounting. We find this step undesirable when handling a large number of sections because the sections need to be washed separately which is very time consuming. Also, on occasion, the gelatin becomes too hard and this causes difficulty when mounting. Sections will keep up to one week in water at refrigerator temperature. Before attempting to mount sections the excess gelatin is trimmed from around the edges and any loose blood colts are removed from the large blood vessels.

For our purpose mounts are made of a uniform size which is 9¼ inches by 12 inches. This size will accommodate the largest specimen which is practicable to cut. The sections are mounted on Whatman number one filter paper 11 by 13 inches which allows for later trimming to the above size. Three sheets of paper are required for each section. The identification and diagnosis of the case are typed on the lower end of the rough side of one of these sheets. The sections are mounted in the following manner. A sheet of plexiglas 11 x 13 inches and 3/16 inches thick is carefully washed and dried and the following freshly made mounting medium, which is kept melted in an oven at 55° to 60° C is poured on it and spread to a thin, even layer.

Gelatin	75 gms
Glycerin	70 cc
10% Sol. of Camphor in Methyl Alcohol	10 cc
Water	850 cc

About 200 cc of mounting medium is required for each mount.

The section, which has been brought from the refrigerator and allowed to warm almost to room temperature, is quickly transferred to the plexiglas plate. This can be readily accomplished by lifting it at the upper two-thirds of the section with the ends of the fingers of both hands. The lower one-third of the section is allowed to contact the plexiglas and the fingers are rolled out from the upper portion. The wrinkles are teased out by use of a bent glass needle such as used in handling quick sections. If any difficulty is encountered, the addition of more mounting medium will help. Care should be taken to orient the section on the plexiglas in such a manner that one would view the finished mount as one would an x-ray film of the specimen in situ. When the section is properly in place an excess of mounting medium is carefully poured over it. Three sheets of filter paper as prepared above are then applied and further mounting medium is flooded over them. This should be sufficient to saturate them. Air bubbles will be trapped between the sheets of paper but the excess mounting medium makes it possible to roll these out with a six inch rubber print roller. Only the weight of the roller should be used until all air bubbles are out and then light pressure may be applied to remove surplus solution. This preparation is allowed to dry thoroughly. The paper with the section adhered to it and covered with a thin film of gelatin can then be stripped from the plexiglas. It will be found that the three sheets of paper and the section have become intimately and permanently adherent to each other and appear as one unit.

The mounts are then trimmed to the desired size and placed between sheets of cardboard and pressed for 24 hours to alleviate their tendency to curl. Care should be exercised in handling the mounts as fingerprints are readily left on the coating of gelatin. To make a more satisfactory permanent mount these sections are sprayed with a very thin coat of clear plastic. Plastic spray in aerosol cans such as is used to spray the chrome on cars has been found satisfactory. The plastic coating is allowed to dry overnight. Mounts prepared in this manner will not show fingerprints and may be wiped with a damp cloth without causing any damage.

• ANGUS D. CAMPBELL, B.A.Sc., M.D., P.Eng.
Director, McIntyre Research Foundation
Toronto, Ontario

JOHANNESBURG PNEUMOCONIOSIS CONFERENCE

The Johannesburg Pneumoconiosis Conference 1959 was convened by the Union of South Africa's Council for Scientific and Industrial Research. It had the active support of the government and the Mining Groups Chamber of Mines. The previous Conference was held 29 years ago.

The Conference was intended "to bring together persons representative of the several scientific disciplines concerned in the study of pneumoconiosis, to discuss recently acquired knowledge and to recommend subjects in which further research is important and likely to be rewarding."

The continuance of new certifications of pneumoconiosis throughout the world and an increasinging number of certifications of new cases of silicosis in South Africa, has brought new activity in studying the pneumoconioses.

Statistical reports of the studies are contained in the latest available reports of the Silicosis Medical Bureau. The Bureau's Report for the two years 1951/52 and 1952/53, and the Bureau's Summary Report as contained in the *Department of Mines Report for 1955* (Table I).

The decided decrease in the number of new cases of silicosis from 1917-1920, when the average yearly incidence of new cases of silicosis and silicosis with tuberculosis was 23.81 per 1000 employees, to a low of 7.10 per 1000 in 1937/38, was quite remarkable, although the increase in number of men employed from 14,000 men to 28,000 rather over-emphasized the decrease, the new cases coming from the older employees.

The decrease correctly was ascribed to medical selection of miners commencing in 1916 and to improvement in mining conditions — wet drilling, dust control, ventilation, and other health protection measures such as removal of tuberculosis cases.

In addition there was controversial compensation certifications in 1953 and 1954 of a disease pulmonary disability, stated to be caused by dust. Certifications regarding that were:

	1953/54	1954/55
White Miners	368	637
Native Laborers	—	285

The disease is omitted from the 1956 Act.

The matter of the numerous certifications for those two years, the latest on which statistics were available, was the matter of much discussion at the Conference.

The partial statistics of the number and incidence of cases among Native Laborers is also significant as per Table II. Incidence is on the basis of 300,000 employed though the average

TABLE I.

Union of South Africa

Comparative Data Regarding the Incidence of Silicosis, Tuberculosis, with Silicosis and Simple Tuberculosis, Amongst Working (White) Miners Examined — Excluding Silicotics Previously Examined.

* Period	Number of Working Miners	Silicosis			Tuberculosis with Silicosis		Tuberculosis		Total
Each Year 1917 to 1955 are in Mines Report	Excluding Silicotics Previously Examined	Per Year Number of Cases	Production Rate Per 1000	Average Age in Years When First Detected	Per Year Number of Cases	Production Rate Per 1000	Per Year Number of Cases	Production Rate Per 1000	Production Rate Per 1000
1917/20	14,070	300	21.32	40.0	35	2.49	37	2.63	26.44
1927/28	14,582	283	19.41	43.0	—	0.00	32	2.19	21.60
1935/36	24,275	221	9.10	49.8	—	0.00	22	0.91	10.01
1937/38	28,174	198	7.03	50.3	2	0.07	31	1.10	8.20
Average of 10 Yrs. 1943/52	Calculated from Table II.		8.65		1	0.04	20	0.76	9.45
1952/53	29,135	243	8.34	52.8	1	0.03	38	1.30	9.67
Rated and Compensation Allowed and with Many Earlier Ratings Certified that might have been "Certified" earlier. Certifications from 30,000 Working Miners were:									
1953/54	30,000 Working	887	29.60	54.2	9	0.30	43	1.43	31.33
1954/55	30,000 Working	418	13.93	54.0	3	0.10	27	.90	14.93

*Data summarized from latest available Reports of the Silicosis Medical Bureau's Report to March 31, 1953 and Bureau's Summary Report in the 1955 Report of the Department of Mines.

at work is reported as 214,662. The native laborers examined are those referred to the Bureau as suspects by mining group medical staffs who now give radiological studies to all natives.

Despite the concentrated attention given to the silicosis problem by medical and mining personnel and the almost fabulous amounts of money spent on medical measures and on engineering controls by ventilation and dust controls that make mining possible, silicosis and complicating tuberculosis were not yet under effective control.

Research into the nature, cause, effect, and prevention of silicosis was stepped up in South Africa to almost a "crash" program.

Members and observers visited the impressive medical and research laboratories of the pneumoconiosis research unit. There, in commodious, well equipped laboratories, animal experiments and chemical and pathological research were in progress and were shown and explained to the visitors. This gave the visitor an intelligent background for conference papers later presented. The lung post-mortem pathological investigation underway and the amount of material involved, was almost incredible to this observer. Lung autopsies on white miners and native laborers who had died from various causes, but for whom there might be compensation claims, were being done at the rate of 16 a day, four days a week.

The dust and ventilation laboratories of the chamber of mines were later visited by the dust and engineering group of the conference. This observer also saw the chamber's centralized laboratories for radioactivity tests regarding their uranium mining.

With these laboratories, and the extensive research center of the S. A. Council for Scientific and Industrial Research seen later at Scientia near Pretoria, the research facilities and personnel undoubtedly represent the greatest concentration of research on pneumoconiosis to be found in the world.

Conference Sessions

The first papers were on silica as the cause of silicosis, the chief pneumoconiosis with which South Africa is concerned.

Nagelschmidt*, for King** and himself, stated their conclusions on the subject of "The Physical and Chemical Properties of Silica, Silicates and Modified Forms of these in Relation to Pathogenic Effects." Nagelschmidt stated: "Free silica, in all its forms tested, was fibrogenic to rats' lungs. Quartz was the most frequent cause of silicosis in man. The three factors that controlled the speed and intensity of the reaction were (1) the order of crystal structure, (2) the state of surfaces, and (3) the size of quartz particles.

"In crystal structure the order of damage to lungs was tridomite, crystobalite, quartz, and vitreous silica."

From laboratory work, chemically and with rats apparently by injections, they found: "In quartz particles of the order of one micron diameter the degree of crystalinity decreases from the center to the surface. The solubility of the surface layers in water or salt solution is higher than that of the interior but etched (with acid) quartz stripped of its outer layer was found to be more fibrogenic than unetched quartz in rats' lungs and more toxic to phagocytes in cell cultures."

Nagelschmidt spoke regarding injected particles of mixed quartz and cristobalite in the range of 0.2 to 10 microns: "The amount of fibrosis was related to surface rather than to mass and when equal surfaces were injected, particles between 0.2 and 2 microns were more fibrogenic than either larger or smaller particles."

Goldstein*** reported that silica dusts of different size range, but equal total surface area, produced the same degree of fibrosis except that the one to two micron particles appeared to be more fibrogenic. That, he found, from quartz injection experiments with two groups of rats. Equal surfaces of each, not equal numbers of particles gave equal fibrosis production in the rats.

TABLE II.
NUMBER AND INCIDENCE OF SILICOSIS AND TUBERCULOSIS
NATIVE LABOURERS DURING THE YEARS 1954/55 and 1952/53
*Bureau Figures from Reports

Category During 12 Months 1954-55	Number Examined by Bureau Living & Deceased	Silicosis		Silicosis & Tuberculosis		Tuberculosis		Total Pulmonary	
		Number	Per 1000	Number	Per 1000	Number	Per 1000	Number	Per 1000
Employed	*4,080	* 819	2.73	*311	1.04	*1,210	4.03	2,340	7.80
No Longer Employed	*2,164	756	2.52	528	1.76	561	1.87	1,845	6.15
Totals	*6,244	*1,575	5.25	*839	2.80	*1,771	5.90	4,185	13.95
1952-1953 Employed and Unemployed on Above Basis									
	*3,040	* 966	3.22	*288	0.96	* 871	2.90	2,125	7.08

*NAGELSCHMIDT, G., PH.D., Head of Pneumoconiosis Group, Safety of Mines, Establishment of the Ministry of Power, Sheffield, England.

**KING, E. J., M.A., PH.D., D.SC., F.RI.C., Professor, London University Postgraduate Medical School.

***GOLDSTEIN, B., M.B., B.CH., Pneumoconiosis Research Unit.

Vorwald* presented somewhat different evidence. He stated, "Particles above three microns were relatively inert. Particles between three and 0.5 microns in size produce lesions of simple nodular silicosis; particles less than 0.5 microns provoke a diffuse reaction: submicroscopic particles provoke a profound production of collagen and fibrosis." He stated the nodular silicosis was less severe than the diffuse type. The lung damage that he quoted in submicroscopic particles of silica appeared in heavy concentrations of metallurgical fume. He named aluminum dust as the only agent which would retard the action of silica.

He stressed the great importance of atmospheric pollutants at the breathing level of the worker. This is the important point in mining.

Hatch** noted the comparatively small amount of silica retained in the lungs. Particles in the submicron range may penetrate the deeper alveolar spaces that mass for mass the finer particles produce pneumoconioses to as great an extent as larger ones.

Gough, the Session Chairman, stated: "We do not know the effect of the very fine particle (submicroscopic). It is theoretical."

It would seem largely theoretical and speculative so far as a miner is concerned. He gets all sizes. With ventilation and dust control the fines will go with the rest except perhaps in air filtered for reuse.

*VORWALD, A. J., B.A., D.PH., D.Sc., D.LL., M.D., Professor and Chairman, Department of Industrial Medicine and Hygiene, Wayne State University, Detroit, Michigan.

**HATCH, T. F., B.Sc., S.M., Professor of Industrial Health Engineering, Graduate School University of Pittsburgh, Pa., U.S.A.

Estimation of Dust in the Mines

The General Session had information on the sampling and estimating methods used at the mines from Beadle***, "Methods of Relating Information Concerned Dust with Medical Criteria." The objective was to determine the average amount of dust breathed at work by nine separate underground occupational groups and correlate that with the incidence and progression of dust lung disease.

Sampling was done over the whole of more than 100 shifts for each of the groups and by the following methods: (a) The Thermal Precipitator (T.P.); (b) The Modified Thermal Precipitator (M.T.P.); and (c) Konimeter (Table III).

Noteworthy is the great reduction in T. P. and M. T. P. counts made by acid treatment; from 430 P.P.C.C. T.P. Count to 121 P.P.C.C., a reduction 72%. Remarkable too is the coincidence of the grand average of counts, 121 P.P.C.C. by T.P. and 120 P.P.C.C. by Konimeter, both after acid. Remarkably close, remarkably low. However, counts were averaged over the whole shift and dust counting samples were taken "under normal conditions" which conditions, it was frankly stated at the Dust Sessions,

***BEADLE, D. G., M.Sc., Ass't. to Group Ventilation Engineer, Rand Mines Ltd.

TABLE III.

1958-59 South African Gold Mining Project — Occupational Ranking by Dust Danger. Summary from Chart Conference Paper 2.3 of D. G. Beadle, M.Sc., Rand Mines Group. Ranking 9 Occupational Groups for Dust Exposure by Sampling Methods and Conditions Shown. Rating "1" Highest Dust Counts. Rating "9" Lowest Dust Counts.

SAMPLES AND COUNTS BY										
STANDARD THERMAL PRECIPITATOR RANKINGS 1 TO 9 BY METHODS BELOW						MODIFIED THERMAL PRECIPITATOR		KONIMETER		AVERAGE MINE METHOD
Average Counts P.P.C.C.		Average Surface Area Square Microns Per C.C.				Average Photo-Electric Readings Arbitrary Units		Average Count P.P.C.C.		
All Particles		All Particles		Particles Minus 5 Microns		All Particles		All Particles		Ranking Concensus
Before Acid	After Acid	Before Acid	After Acid	Before Acid	After Acid	Before Acid	After Acid	Before Acid	After Acid	by A.D.C.
1	1	1	1	1	1	1	1		2	1.11
2	4	2	6	3	4	4	6		7	4.22
3	2	2	3	2	3	5	2		5	3.00
5	3	4	2	4	2	3	7		8	4.22
4	5	5	5	5	5	7	2		4	4.66
7	6	7	7	6	7	9	9		6	7.11
9	8	9	9	9	9	2	7		9	7.89
6	9	8	8	7	8	6	2		1	6.11
8	7	6	4	7	5	8	2		3	5.55
AVERAGE COUNTS										
P.P.C.C.		Square Microns per C.C.				Arbitrary Units		P.P.C.C.		
430	121*	1060	410*	730	280*	39	10*		120**	

*Beadle stated his belief that the count before acid treatment was closest.

**50 Konimeter samples per shift — 91 shifts.

The object of the Project was to compare the Ranking by Dust Danger with Silicosis Production.

The Rankings by Different Methods varied — Silicosis Production by Occupations was not available.

do not always exist in practice. Things do go wrong.

The art of dust sampling was shown to be far from an exact science. The T.P. is a research instrument on which much research remains to be done to interpret its results. Konimetry sampling remains standard in South African gold mines as in Ontario. The M.T.P. is used in S. A. coal mines.

The sampling project did produce some results, but the statisticians working on the medical records had so far failed to produce silicosis production and progression results of the occupational groups.

The statistical study above, was described by Wiid* as "Statistical Studies of Pneumoconiosis Related to Dust Exposure, Occupation and Radiological Category, Progression and Other Relevant Factors."

The study was in retrospect of all white (European) miners first certified as suffering from silicosis during the period 1946-1956. The relevant data was obtained on 3,800 of the ± 4,500 miners certified in the time. Half of those would have been certified in the last three years of the period. Little progression of these could be expected since then. No progressions were reported to guide Conference discussions as to whether the controversial early nondisability certifications were actual silicosis cases that would progress to disability.

The study reported only on the time spent in mine service and in broken time by the cases and on adiposity determinations.

Adiposity of the 3800 miners were determined from their annual examinations according to the deduced formula:

$$A = 0.3107 \text{ T.B.M.} - (.00009603H^3 - 1905)$$

where A = adiposity; T.B.M. = total body mass in pounds

H = height in inches.

Adiposity studies on broken time versus continuous service and change in adiposity with increasing service were continuing and may later throw light on progression.

Table IV summarizes the latest available dust counts for the year 1955. Samples taken are under "normal working conditions." Many other samples are taken at dusty areas for corrective measures.

11,240 sample spots taken at all underground working points, including headings and stopes (160).

22,387 sample spots from all underground averaged 130 P.P.C.C.

1,680 sample spots from surface averaged 150 P.P.C.C.

*Wiid, A. J. B., D.Sc., (Pretoria). Head of Statistical Division. National Physical Laboratory, C.S.I.R.

TABLE IV.

SUMMARY OF VENTILATING AND DUST COUNTS AT GOLD MINES FROM THE MINING ENGINEER'S REPORT, UNION OF SOUTH AFRICA YEAR ENDING SEPT., 30, 1955 — GOLD MINING

	1954	1955
Daily C.F.M. Down Cast Air	31,522,500	34,605,500
Maximum Number of Persons Underground at One Time	220,174	237,323
Air Per Person C.F.M.	143	145

DUST COUNTS KONIMETER P.P.C.C.*	Number Sample Spots	P.P.C.C. Average Count
Intake Air at Surface Down Cast Shafts	308	40
Intake Air Leaving Down Cast Shafts	544	60
Working Points		
Stopes	5,688	160
Development Ends	3,200	152
Shaft Sinking	172	146
Other Points	2,180	
TOTAL WORKING POINTS	11,240	160
Return Air Upcast Shafts to Surface	156	70
Other Returns from Working Points	4,919	120
Other — Miscellaneous	6,072	
TOTAL UNDERGROUND	22,387	130
SURFACE DUST SAMPLING		
Working Points		
Assay Offices	36	110
Metal Working Shops	200	132
Carpenter Shops	16	22
Welding Cubicles	128	320
Reduction Works	1,083	150
Miscellaneous	145	70
TOTAL SURFACE	1,608	150

*Particles Per Cubic Centimeter.

Types of South African Silicosis

Webster** in his paper, "The Pathology of Gold Miners' Silicosis," reviewed the changes in silicosis in South Africa and the recent pathological findings of lung autopses of 1,835 mine workers who had died from various causes.

Webster reported silicosis lesions found among 532 of the 835 white miners and among 209 of the 1,000 native laborers as per Table V. "The proportions had nothing to do with prevalence." The classifications were not correlated with findings in life and no information given as to the number certified for compensation benefits which are allowed for pneumoconiosis of the cardiorespiratory organs (by whatever means discovered) which has been caused by exposure to dust in the course of work in a dusty atmosphere in "registered" mines.

The average of the whites increased from 55.7 years (18.4 years of service) for the "massive fibrosis" rating, showing that "the more severe the lesions the older the age group."

Tuberculosis

King's paper, "The Experimental Infective Tuberculosis," presented by Nagelschmidt, stated

**Webster, I., M.B., B.Ch., B.Sc., (Hons.) Pathologist, Sub-Director, Pneumoconiosis Research Unit.

TABLE V.

Particular Silicotic Lesions Found	White Miners		Natives — Bantu	
	Total Number	Nmber with T.B.	Total Number	Number with T.B.
Dust Reticulination — DR	63	2	63	20
Dust Collagenisation — Collag.	90	6	52	23
Occasional Islets (1 or 2) — Occ.	77	7	16	6
Few Islets (3-15) — Few	141	18	55	12
Moderate Number of Islets (15-20) — Mod.	96	18	13	4
Large Number of Islets (more than 30) — L.Mo.	30	4	—	—
Massive Fibrosis (Focus of Pigmented Fibrosis More than 2.4 Cms. in Dia.) — M.F.	27	6	10	2
Total Number	532	60	209	67
Average Age at Death	60.0		47.3	

that animal experiments using dead and living B.C.G. produced more serious and larger lesions than produced by free quartz or kaolin and persisted longer although there was regression after about a year. Similar results were obtained with coal and dust and B.C.G. — both killed and living — The Wells vole bacillus, two human strains of M tuberculosis (3073 and B) and a photochromogenic mycobacterium. All animals inoculated with 3073 died. They showed extensive and severe lesions in the lungs. The experiments continue.

King concluded that the experiments (animal) lend support to the belief that the progressive massive fibrosis of coal miners, kaolin workers, and perhaps others, always develops on a background of simple pneumoconiosis but is really due to an infective process "modified in a dusty lung — in the direction of increased fibrosis."

Gough* "Pneumoconiosis and Infection" stated: "Men exposed to coal dust do not appear to suffer unduly from tuberculosis. This is contrary to men exposed to highly silicosis dusts. Nevertheless, progressive massive fibrosis in coal workers is believed to be due to the combined action of dust and tuberculosis. If massive fibrosis is an infective form of pneumoconiosis there seems no satisfactory alternative to the tuberculosis hypothesis."

Meiklejohn** in "The Clinical and Epidemiological Aspects of the Role of Tuberculosis in Pneumoconiosis," concluded: "Silicosis specifically predisposes to infection with the tubercle bacillus. The soil — the lung tissues — is altered so that the growth of the organism is facilitated. It must be emphasized that it is not necessary for identifiable silicotic nodules to be present; the mere deposition of particles of silicon dioxide (silica-quartz) suffices. In the diagnosis of silicosis you should always be expecting tuberculosis."

All did not agree that tuberculosis was always the infection that helped produce massive fibrosis. Professor (Dr.) Pendergrass*** in discussion showed a number of lung films. Several showed massive fibrosis which he stated were not due to secondary tubercular infection.

Hope in treatment of tuberculosis in silicotic miners was reported by Pringle† in "The Treatment of Tuberculosis Associated with Silicosis." He reviewed the results of sanitarium treatments with standard methods aided by the new drugs, Streptomycin with P.A.S. and/or I.N.H., among three groups:

I. Miners suffering from silicosis and tuberculosis — 91 patients.

II. Miners suffering from tuberculosis only — 65 patients.

III. Nonminers with tuberculosis only — 27 patients.

He found that while deaths among Group I were somewhat higher than among the other two groups, this appeared to be due to their significantly higher average age; 64 as compared with 53 and 58 in the other groups.

Of the surviving members of Group I, 39 are "well and working"; 16 are "well and retired"; four remain in hospital with other major ailments and two are at home "not well."

Dr. Pringle concluded: "The results of treatment of tuberculosis in association with silicosis are encouraging and compare favorably with the results obtained in cases suffering from tuberculosis only."

Prevention of Tuberculosis in Silicosis Cases

Sluis-Cremer†† in "The Therapy of Silicosis" reported on a project of the Miner's Chest Clinic regarding preventing tuberculosis from developing among silicosis cases.

A series of such cases with no suggestion of open or active tuberculosis had been treated six months or longer before with tuberculostatic drugs. Results were being assessed clinically and physiologically with comparisons to be made with a similar untreated control group.

Statistical proof regarding hoped for prevention of the development of tuberculosis among silicosis cases where such development might occur, would be welcome news. Such proof one way or the other should be available soon.

*GOUGH, J., B.Sc., Professor of Pathology, Welsh National School of Medicine, Cardiff, Wales.

**MEIKLEJOHN, A., M.D., M.R.C.P., F.R.F.P.S., F.R.S.E., D.P.H., Lecturer in Industrial Health, University of Glasgow.

***PENDERGRASS, E. P., M.D., Professor, Head of Radiology Department, Hospital of the University of Pennsylvania, Philadelphia, Pa.

†PRINGLE, M. A., M.B., CH.B., Medical Superintendent, Springkell Sanatorium.

††SLUIS-CREMER, G. K., M.D., Physician in Charge, Chest Clinic, Pneumoconiosis Research Unit.

Prevention of Silicosis

The immunological approach, on the basis of antibodies developing in the lungs to prevent silicosis from developing, was described as a promising new approach to the explanation of individual susceptibility. The hope appeared to be that if the presence of antigen antibodies could be proven the antibodies could be stimulated and act in prevention. To this observer it seemed somewhat like helping the good guys on our television dramas to beat the bad guys.

Webster presented a paper on the subject on behalf of his group of seven research workers from various scientific disciplines. He stated that in Hungary in 1956 Kalman had demonstrated that silicotic rat serum produced lesions, but had been unable to demonstrate any specific antibody to counteract it.

Webster reported progress in finding that local and circulating antibodies develop in lungs and may prevent silicosis. If the theory is a valid one, antibodies should be found in miners with severe silicosis, but difficulty there was in excluding tuberculosis. He concluded: "There is a basis for the Immunological Approach to Silicosis."

Vigliani* contributed discussion along the same lines. He stated that the approach offered a better explanation of the pathogenesis of silicosis than did the solubility of silica dust or the "mechanical" or "surface activity" hypotheses. His statement that "silica modifies lung cells in such a way that they set free the substances that continue silicosis development" seems to bring the approach right back to some form of silica activity.

Chemical Approaches

Studies of the chemical changes qualitatively and quantitatively which occurred in experimental animals dusted with quartz were being made. Progress reports were made of the findings on behalf of the six chemists, who, with one veterinary and one medical doctor, made up the research team. Various members discussed mucopolysaccharides, lipids, amino acids and the histochemistry of silicotic islet formation.

"The Chemical Approach to Therapy of Pneumoconiosis" by Sutton**, chief chemist of the above chemical research project of the pneumoconiosis unit, described the approaches made in the above noted chemical research, to the problem of prevention of silicosis.

Sutton stated, "The aluminum therapy of Denny, Robson and Irwin*** was based upon the principle that silica could be rendered innocuous by means of a protective layer. The effectiveness of aluminum hydroxide against silica toxicity has been demonstrated recently by Marks who has also shown that 'Compound 48/80' a water soluble polyamine is about equally effective and probably forms a protective layer on silica since it reduces solubility." In his paper he stated "48/80 cannot be considered for therapeutic use because it is a very powerful histamine releaser. Other agents investigated had little or no silica antiactivity."

Dr. Sutton stated two approaches to prevention were open and investigated, as follows:

1. "Dissolved silica being, as he said, generally believed to pass harmlessly from the system, silica particles might be dissolved out of the lungs by some agent."
2. "There was the prospect of coating the surfaces of quartz dusts in vivo to convert them to inert dusts and thus to lessen or abolish their fibrogenicity."

Sutton gave a thorough trial to number one, but after many avenues of approach had failed to produce results the investigations were dropped.

"There remained, he stated, 'proposal two — the prospect of coating the surfaces of quartz dust in vivo to convert them into 'inert' dusts and thus to lessen or abolish fibrogenicity." For that, he resorted for coating to the use of the chelating agent aluminum (EDTA). His choice of aluminum, he states, was because of its place in the stability series (regarding chelating) and because of its previous application by Denny, Robson, and Irwin in aluminum therapy. Regrettably he did not use the aluminum directly as they did.

As further proof of his lack of aluminum coating, he states: "We were unable to detect any difference in lung pathology between guinea pigs which had inhaled quartz whether they had been injected with single doses of the aluminum or not."

Denny, Robson, and Irwin, in their paper referred to by Sutton, show conclusively that they could place directly a thin impermeable coating of aluminum on quartz surfaces in the test tube. That coating prevented the solution of silica and proved that the damaging surface action of silica was prevented. They showed too that injections of a mixture of aluminum and quartz into animal tissues did prevent fibrosis in those tissues.

In order to be effectual there must be an intimate mixture of the silica with its neutral-

*VIGLIANI, E. C., M.D., Director of Occupational Clinic "Luigi de Voto," Milan, Italy.

**SUTTON, D. A., B.Sc., (Hons.), A.R.C.S. (Hons.), PH.D., Chief Chemist Pneumoconiosis Research Unit.

***DENNY, J. J., ROBSON, W. D. and IRWIN, D. A., Prevention of Silicosis by Metallic Aluminum, *Canadian Med. Association Journal*, 1937, 37:1.

izing agent aluminum. Feeding aluminum by mouth, by intraperitoneal injections and by the subcutaneous route will not prevent lung damage by inhaled silica.

During, and immediately following, the Conference some South African mining and medical people noted that their research to find a chemical agent to aid in the prevention of silicosis had lead inevitably to aluminum.

Some were taking a new look at their continued production of silicosis; why aluminum has not been used in South Africa and the place it might have in their prevention methods.

They stated South Africa is ideal for a controlled trial of aluminum prevention of silicosis. They have been using known effective medical and engineering methods for years. In that country there are the most uniform quartz ores, mining conditions, medical controls, and Federal jurisdiction of any mining industry. With employees numbering hundreds of thousands exposed to silica (quartz) dust, they have many silicosis cases at all of their mines which have been operated for many years. Production and progression records among white miners are known and kept. The benefits of the use of aluminum could be determined by using one or more mines as controls; while at other mines an equal number of white miners would receive McIntyre Aluminum Powder group treatments under Medical supervision according to standard 'inhalation' methods.

From results in Ontario generally, and particularly at McIntyre-Porcupine Mines in the use of aluminum as an aid to the basic medical and dust control prevention methods, this writer, who has kept the available data on every known case of silicosis at McIntyre's 1000-miner mine, has no doubt that results in South Africa would be favorable and in a very few years.

Drastic reductions since 1944 in the number of new cases of earliest nondisability silicosis and in the number of cases recognized for disability compensation are shown in the Sellers-Wheeler paper in this Journal, "Recent Trends in Silicosis Among Miners in Ontario."

The statistics include all Ontario miners working and nonworking, but do not differentiate between those who received aluminum treatments and those who did not.

Most encouraging is the fact that only two cases of compensable silicosis were assigned to McIntyre in each of the years 1958 and 1959. The two of 1958 left McIntyre in 1946; 12 years before. The two of 1959 left in 1943 and 1945; 16 and 14 years before. All had only minimum aluminum treatments commencing 1944. None of the four had even earliest silicosis ratings on leaving McIntyre.

The Conference served its purpose in bringing together persons of scientific disciplines concerned in the study of pneumoconiosis. Much acquired knowledge was discussed and many subjects recommended for further research.

Of first importance is the final recommendations of the Conference (Appendix): "That more effective liasion and cooperation should be established between the various disciplines involved in study and control of pneumoconiosis."

APPENDIX

As a result of its deliberations, the Conference adopted the following:

I. In the Field of Etiology and Pathology

IT IS RECOMMENDED:

1. That as observations on man with regard to the pathogenicity of silicates are incomplete, it is desirable that further observations on man and experiments on animals be carried out.

2. That the effects of the inhalation of Group III elements, including the rare earths, singly and in such combinations as they occur in industry, should be investigated.

3. That the importance of submicroscopic particles in the pathogenesis of pneumoconioses should be intensively investigated.

4. That it is desirable that pulmonary dust retention studies be carried out on subjects with abnormal conditions of the upper respiratory tract and the lungs, and that studies of clearance of dust from normal and impaired lungs should be continued and extended. Sites of deposition of dust within the pulmonary lobule in relation to particle size above and below 0.4 microns, and the pathological significance of site of deposition also require study.

5. That clearer demonstration of pathological anatomy can be obtained by cutting Gough and Wentworth sections from expanded lungs. This technique should be used routinely, wherever possible, especially where radiological and pulmonary function intercorrelations are to be made with pathological anatomy.

6. That (a) the pathological studies of the bronchi in South Africa gold miners should be extended, with special reference to changes in the bronchioles, and (b) acute and chronic effects of dust inhalation on the respiratory system should be investigated in persons employed in dusty occupations, irrespective of the presence or absence of pneumoconiosis.

7. That a uniform definition of the condition chronic bronchitis be established, and that a comparative epidemiological study of this condition in dusty and nondusty occupations be undertaken.

8. That the prevalence of the several types and degrees of emphysema should be studied more extensively, comparing their incidence in dusty and nondusty occupations and in various age groups.

9. That as atrophic conditions have been found in the heart in certain cases of pneumoconiosis, including Rand gold miner's silicosis, further studies of this condition should be carried out.

10. That correlation of radiological and pathological changes should be made on as many and as representative samples as possible, to provide additional information on (a) early diagnosis of pneumoconiosis, and (b) the differentiation of the radiological appearance of pneumoconiosis from those of other conditions. (Paper mounted whole lung sections should be filed with the corresponding radiographs for easy reference and research).

11. That the size, mineral type, concentration and pulmonary retention of dust particles responsible for asbestosis should be further investigated.

12. That the relationship between exposure to asbestos and mesothelioma of the pleura, which appears more prevalent in South Africa, should be further investigated. This study would be facilitated by the establishment of a central registry of cases under the South African Council for Scientific and Industrial Research.

13. That the causes of massive pneumoconiosis occurring in occupations in which there is no obvious free silica hazard should be examined, especially from the point of view of the quantity and composition of dust in the lung, the relative importance of quartz, coal, and other dusts, and the presence of infection.

14. That the pathological criteria whereby lesions due to dust alone, due to tuberculosis alone, and due to the interaction of both, should be clarified by further research.

15. That as the immunological approach to silicosis and other pneumoconioses appears to be a promising line of investigation, research in this field should be intensified.

16. That investigation be undertaken into the pathogenesis of rheumatoid pneumoconiosis (Caplan's syndrome) and whether this syndrome occurs in gold miners.

17. That chemotherapeutic and antibiotic approaches to the prevention of the progression of pneumoconiosis should be further investigated.

18. That agreement should be reached on the usage of the terms reticulin, collagen, and hyaline.

19. That study of the biochemical changes in experimental silicosis should be extended to include all stages of this condition.

II. In the Field of Physiology, Radiology, and Clinical Medicine

IT IS RECOMMENDED:

1. That in view of the fact that asbestosis is an important industrial problem it should be subjected to intensified epidemiological, pathological, clinical, physiological, radiological and immunological studies in both occupational, and nonoccupational exposures.

2. That in any prospective study relating dust exposure and pneumoconiosis, and investigation of the prevalence and attack rate of tuberculosis should be included.

3. That further epidemiological studies be instituted on representative samples of South African mining and nonmining populations and of the correlation of symptoms, of pulmonary function loss, x-ray categories, dust exposure and other relevant factors such as smoking.

4. That a study be made of factors affecting the progression of early pneumoconiosis and pulmonary disability, especially of the effect of continuing work in dusty occupations.

5. That the possible relationship between scleroderma and occupation should be further investigated.

6. That studies of cardiopulmonary function be conducted on persons employed in dusty occupations and on suitable control groups, to ascertain the relationship between cardiopulmonary disfunction and occupational history, clinical and radiological findings.

III. In the Field of Dust and Engineering

IT IS RECOMMENDED:

1. That measurements of dust in pneumoconiosis studies should relate to the respirable fraction of the dust cloud, this fraction being defined by a sampling efficiency curve which depends on the falling velocity of the particles and which passes through the following points: effectively 100% efficiency at one micron and below, 50% efficiency at five microns, and zero efficiency for particles of seven microns and upwards; all the sizes refer to equivalent diameters. (The "equivalent diameter" of a particle is the diameter of a spherical particle of unit density having the same falling velocity in air as the particle in question).

2. That in the light of present knowledge, dust measurements to assess the health hazard should be expressed as the average level of dustiness over an appropriate period of sampling, such as a shift. This measurement may be made by averaging a number of samples, or by using an instrument which automatically averages the dust over the period. Exceptional peaks of dust concentration might also be recorded.

3. That in the light of present available evidence, the best single descriptive parameter to measure be considered (a) in the case of coal dust to be the mass concentration of the respirable dust, (b) in the case of quartz dust to be the surface area of the respirable dust.

4. That more use should be made of dust sampling instruments, with size selecting devices, which collect only the respirable fraction of the dust, as defined in paragraph I.

5. That wider use be made of long period dust sampling instruments.

6. That more attention should be paid to designing correct dust sampling strategy, bearing in mind the difference between sampling for purposes of dust control and sampling in order to determine the health hazard.

7. That the variations in dust concentration should be studied both during single shifts and by comparison of different shifts.

8. That further studies should be made of the quantity and character of the dust produced by various mining operations.

9. That studies should continue on the build-up and decay of dust concentrations under mining conditions.

10. That further studies should be conducted to ascertain the dust exposure in various occupational groups.

11. That epidemological studies to determine that dose-response relationship in man should be continued and expanded.

12. That individual cumulative dust exposure records should be kept for men working in dusty occupations. In particular it is recommended that a start be made on South African gold mines by keeping special records of a sample of new entrants, showing their hours underground and their dust exposures based on random samples covering a full shift. Their medical data should be especially recorded.

13. That further studies should be made of possible individual variations in susceptibility to dust. (Note: The records obtained from recommendation 12 may give some information on this subject).

14. That further studies should be made of pulmonary retention of particles of above two microns equivalent diameter.

15. That detailed studies should be made of the size distribution, composition and amount of dust found in the lung tissue of miners with and without pneumoconiosis. Where possible these measurements should be related to dust exposure records and x-ray appearances.

16. That the composition of the submicroscopic particles which occur in mines and dusty industries should be studied in more detail.

17. That further animal inhalation experiments should be made with freshly produced dusts typical of underground mine dusts. These should be designed to produce more evidence of the effects of fine dust (of the order of one micron and below) as compared with coarser dusts as they occur in mines.

18. That further animal inhalation experiments should be carried out to study the effect of salt aerosols on pneumoconiosis.

19. That studies be made of the size distribution, composition, and amount of dust found in the lung tissues of animals which have inhaled dust of known size distribution.

20. That it is desirable to assess the degree of pathogenicity of various dust mixtures with variable amounts of free silica.

21. That experiments should be conducted to determine the effects of ionising radiations on pneumoconiosis.

22. That studies should be conducted to determine the effect on pneumoconiosis and pulmonary disability of nitrous fumes and other gases as they occur in mines under similar conditions to which men may be exposed to them, i.e. in the case of nitrous fumes, occasional short exposures.

23. That studies should be conducted in South Africa to determine the effects of fine carbon particles, and particles containing organic salts occurring in South African gold mines.

24. That inasmuch as the reduction of pneumoconiosis depends mainly on dust control, intensive research should be carried out in regard to improved dust suppression measures. In general, such research should include: (a) development of new methods of dust suppression, (b) further studies of the effect of different ventilation methods on dust concentration, (c) further studies of the effect of different dust suppression methods on the concentration and properties of dust, (d) the possible application of local protective equipment to provide workmen with clean air.

25. That more effective liaison and cooperation should be established between the various disciplines involved in the study and control of pneumoconiosis.

PROBLEMS OF SILICOSIS IN BRITISH COLUMBIA

• E. O. T. SIMPSON
Acting President and
Chairman, Committee on Silicosis
Mining Association of British Columbia

British Columbia is one of the oldest mining provinces in Canada. Its mines have been active for more than 100 years. Even under present semidepressed metal-price conditions applying to most of its mineral production, mining continues to rank as the second basic industry in British Columbia, with annual production valued at 150 million dollars or more. Underground mining has been carried on continuously in several areas of the province for periods of 60 years and more. As one consequence, and in common with the provinces of Ontario and Quebec, there was a substantial incidence of silicosis among hardrock miners (and to a lesser degree among coal miners) many years before this disease was officially recognized.

In Ontario, where some 11,000 men were employed in the mining industry at the time, silicosis was made compensable in 1926. It was not, however, until 10 years later, with a total of 14,179 employed in all phases of the mining industry (1926 total was 14,172), that silicosis was made compensable under the industrial diseases schedule of the British Columbia Workmen's Compensation Act. As we shall suggest later, this 10-year delay in official recognition of silicosis may have been an important contributory factor in our present problems.

The Financial Problem

Examination of Workmen's Compensation Board statistics covering the financial operations of class 2 4 (Silicosis — Metal Mining) indicates that during the period 1936 to 1958 inclusive, a total of 798 claims for silicosis compensation have been accepted. Total cost of these claims has been $10,542,666.04. Annual assessments against the mining industry in British Columbia have ranged from a commencing low of $115,000 in 1936 to $684,000 in 1957 and $667,000 in 1958. While statistics for 1959 are not yet available, it has been estimated that silicosis will have cost the mining industry well in excess of $800,000 during the past year. Obviously, such costs are a matter of grave concern to an industry presently employing only about 6,000 men in its lode and coal mines and concentrators.

At the end of 1959, there were 354 pensions being paid, with 179 cases of stage one, and eight cases of stage two silicosis in suspense. This represents over 44% of the total accepted claims since 1936.

Recent tabulations suggest that the financial problems of silicosis compensation are closely linked with legislative changes and the economic well-being of the mineral industries during a given period. The tables covering the period 1936 to 1958 can be divided into the subperiods 1936 to 1941; 1942 to 1950; 1951 to 1958; 1959 onward.

The first period reflects the introduction of legislation to provide compensation for silicosis, with an initially low but rapidly rising number of accepted claims. During the first period, (and for that matter during the first 10 years of silicosis compensation) the fund was unduly taxed by the accumulation of cases from the precompensation years. The initial rate of 2.50 ($2.50 per $100 of dust-exposed payroll) was maintained for only two years, being doubled thereafter from the remainder of the period. The second subperiod began with a further 25% boost in rate to 6.25%. It will also be noted that the numbers of men employed showed only minor variation until 1942 when the impact of the war years was felt. While assessable payrolls remained fairly constant, assessments to meet pension requirements almost quadrupled by the end of the period, proportionately increasing the burden on the industry.

The effect of postwar acceleration of mining activity arising from increased metal prices and demand is shown in the second subperiod, 1942-51, during which the number of employees increased from a low of 6,700 to almost 11,000, providing a much broader base for assessment. In consequence the rate was reduced from a high of 6.25 to 3.25, and to 3.00 in 1952. During this period both costs and the number of accepted claims tended to decrease.

During the third of these arbitrarily designated periods, from 1951 to 1958, the tabulation of numbers employed shows the sharp decline from more than 11,000 to 5,400. Falling base metal prices and the rapid erosion of the coal mining industry owing to competition from oil and natural gas combined to reduce the numbers employed to the lowest levels in a quarter of a century.

Concurrently, amendments to Workmen's Compensation legislation substantially increased and broadened benefits and thereby compensation costs as the social service concepts of compensation gained ascendency over the insurance concept. The greatly reduced payrolls available for assessment to meet rapidly mounting pension costs inevitably forced up the assessment rates from a comparatively low figure of 3.00% in 1952 to 4.50 in 1956 and 5.62 in 1958. Even this rate of 5.62, highest since 1947, was not sufficient to meet demands on the class 2/4 fund, and the rate was increased to 7.00 in 1959.

During the past year the number employed was approximately 5,500. It is not considered likely that this will increase much beyond the 6,000 mark during 1960. Silicosis compensation costs are apparently still increasing, and there is therefore little if any prospect of a reduction from the 7.00 rate in the immediate future.

The gravity of the problem becomes even more apparent when the total compensation cost for the mining industry is considered. The rate of 7% applies to all dust-exposed personnel. Added to this is a class 4/11 (Accident — Metal Mining) rate of 4¼% which makes an aggregate compensation assessment of $11.25 per $100 of dust-exposed payroll in this province. It has been repeatedly emphasized by this Association that these levies against the mining industry are exerting a strong influence against the flow of venture and investment capital into British Columbia, so vital to the maintenance and development of this industry.

As will be noted in more detail later, the mining industry in British Columbia has achieved a substantial measure of success in its efforts to improve ventilation and suppress dust. Most unfortunately, adequate statistical evidence as to the incidence of new cases of silicosis, date of first exposure to dust, and duration of exposure are not available. This is a situation which should be remedied without delay. Examination of a large number of case histories suggests that (with only one or two exceptions) virtually all claims granted have been to claimants whose first exposure to dust was prior to the introduction of silicosis compensation in 1936, and prior to the intensified dust control and ventilation programs of the past 20 years. The mining industry and the Compensation Board are cautiously hopeful that the experience in Ontario as set forth in Dr. Paterson's recent report (Ontario Department of Mines Bulletin 158, *Silicosis in Hardrock Miners in Ontario*) may be repeated in British Columbia, subject perhaps to the effects of the 10-year delay in recognition previously mentioned. If we can tentatively accept for British Columbia his Ontario statistics showing average duration of exposure of almost 23 years; average age of first recognition of almost 63 years; and increase in life expectancy of silicotic miners since 1926 by more than 20 years then the inference may be drawn that dust control and ventilation combined with certain other factors have materially reduced both the incidence and the severity of silicosis. We can only hope that this is indeed the case.

There is compelling evidence to suggest that mining companies operating at the present time have been forced through circumstances beyond their control to assume a disproportionate share of the burden of both silicosis prevention and silicosis compensation. Certainly, many of the case histories of silicotics previously and currently being compensated in British Columbia show that these men were "dusted" in the employ of companies that have long since ceased to exist and, in many instances such companies made no contribution to the silicosis fund. Additionally our records show that virtually all silicosis pensioners were dust-exposed prior to the introduction of legislation providing for silicosis compensation. It is indeed ironic and inequitable that mining companies now starting operations should be required to assume the responsibility of compensating former employees of companies that ceased to exist more than a quarter of a century ago.

As previously noted, this burden becomes even more difficult during periods of reduced mining activity when the numbers of operating companies and the assessable payrolls are curtailed, as has been the case in recent years. At the same time, these companies are properly required to maintain and increase their efforts to improve ventilation and to suppress dust in working places. The statistics indicate, of course, that a good part of the heavy and necessary expenditures for these purposes will represent a substantial economic benefit to the mining companies of 20 years from now. In short, the mining companies of today are compelled to accept responsibility for the sins and omissions of past generations and concurrently to ensure that future generations may reap the benefits of good mining practices, good ventilation and preventive measures.

Our Association is of the opinion that it is both feasible and desirable to provide some measure of financial relief to presently existing mining companies. While we have not yet reached any definite conclusions as to the best method of achieving this objective, there are several ways in which this can be done, including that of relieving them of financial responsibility for silicosis cases they did not create or which were created prior to introduction of compensation legislation. Another possibility which has attracted some support is that annual assessments should be reduced (perhaps by as much as 50%) below the actual cost of providing pensions during a given year and that the pension costs should be amortized over a much longer period. This assumes and it is fully expected that one incidence of new cases during the next 20 years will decline sharply. Existing companies will thus obtain some immediate relief from their present heavy burden. It is realized that companies yet unborn will have to accept part of this spread but we do not consider it in any way unrealistic or inequitable to suggest that part of the burden of compensating silicotics created 25 or 30 years ago should be passed on, subject to the application of sound actuarial advice. Another definite benefit of this proposal would be to offset to some extent the adverse effects of periods of curtailed mining activity and reduced payrolls available for assessment.

The Social Service Problem

In many respects the underlying insurance principle of workmen's compensation is giving way to the social service concept that compensation should be paid regardless of the circumstances. Nowhere is this trend more obvious that in compensation for silicosis. Perhaps this is more markedly so in British Columbia than in other provinces. From introduction of silicosis compensation in 1936, efforts to broaden the scope of legislation and to increase the discretionary powers of administrators have been unremitting and perhaps all too successful.

In a brief presented to members of the Government of British Columbia last year by our Association it was stated that: "While the fibrotic condition to be found in virtually all mine workers is in most cases neither of a disabling nor of an incapacitating nature, and while the lessened capacity for work is in most cases the normal product of advancing years, or the result of unrelated disease, a literal interpretation of Section 8 (5) (the section in the B.C. Workmen's Compensation Act defining silicosis) could offer the ultimate 'reductio ad absurdum' in Workmen's Compensation: Every person who has at any time worked at a dust-exposed occupation in the mining industry of British Columbia, and for whatever reason is able to demonstrate a lessened capacity for work will be able to claim, and presumably will be able to obtain, a silicosis pension."

The definition of silicosis just referred to is identical to, or very similar to, the definition in the Ontario and other Acts. Additionally there appears in the British Columbia Act a discretionary provision which we commonly refer to as the escape clause and which reads in part: "But this provision shall not prevent an allowance by the Board of any case due to silicosis that the Board considers should in justice be allowed."

It is of some interest to note at this point that during 1958, 18 silicosis claims were paid to claimants over 65 years of age; five were paid to claimants over 70; one was paid to a claimant over 75; one was paid to a claimant over 82.

Our contention is that many of these claims were paid without proper consideration of the extent, if any, of disability or lessened capacity for work attributable to silicosis, and that many of them were, in fact, paid as a form of supplementary social service benefit.

Recent experience offers some interesting evidence that uncomplicated silicosis is not by any means as deadly or as serious as once thought, and unless complicated by some infectious or other chest condition has little effect even where shown to a marked degree by x-ray. It seems that there is little or no effect on longevity and in fact, perhaps attributable to extra care, those with silicosis may live longer than the average person. Recent experience in British Columbia points up the lack of effect on longevity. Due to shut-down of one of the older and larger mines, men were laid off and applied for silicosis pensions. In many instances these men had been doing relatively heavy work, such as timbering, blacksmithing, etc., and continued to do so well past the age of 60 (and one up to 75) although examinations after receipt of their applications showed that they were suffering from a marked degree of silicosis. In one case (not in mining) a workman with marked silicosis at age 91 made a claim because in the previous two years his earnings had decreased due to his inability to work full time.

Perhaps research should now be done to determine in the light of recent and present experience the effect of (a) slight, (b) moderate, and (c) marked degrees of silicosis in relation to ability to do (a) heavy work (b) light work (c) other activities. It may be that rules and regula-

tions should be laid down by each province so that Compensation Boards could be consistent in their awards.

It is by no means our intention to convey that the Workmen's Compensation Board in our province is any more or less impartial and capable than those of other provinces. We believe that the B.C. Board has performed its duties well in accordance with existing legislation. Unfortunately it is becoming increasingly apparent that certain segments of the public are unwilling to accept the Board's findings. With increasing frequency, the Board's decisions are being challenged in the courts, questioned by politicians, rejected by union leaders, and made the subject of hysterical headlines in the newspapers. As an aside, it is of interest to note that the medical profession is by no means immune from these evidences of partiality and antagonism. From our own experiences we can concur entirely with Dr. Paterson in his remarks pertaining to "Problems in Diagnosis as they are Related to Compensation." It is not encouraging to read in the newspapers that a member of the profession, whether specialist or self-styled specialist, has publicly challenged the findings of the Board's specialists and referees; has determined that his patient has silicosis, and states that the patient is therefore entitled to a pension. To us this appears to be a ursurpation of the authority of both the Board's medical staff and of the Board itself. We are strongly of the opinion that in so far as is possible, the impartiality and competence of the Board and its staff should not be subjected to public or legal challenges of this nature.

Perhaps the most serious social service aspect of silicosis compensation is that arising from the problems of tuberculosilicosis. Attention was focused on this problem during a Royal Commission in 1951-52. Our Association at that time stated that in its opinion a claimant otherwise qualified to receive silicosis pension should not be deprived of that pension because the otherwise compensable silicosis in his case had become complicated by tuberculosis. Amendment of legislation followed. As one result of the interpretation placed on this change in legislation, it now appears that any claimant, having any degree of silicosis almost automatically receives a pension where tuberculosis is contracted. This interpretation, which we are unofficially advised is somewhat similar to the procedure followed in South Africa, places what we consider to be an unjustified responsibility on the mining industry.

A claimant may have spent only a short period of time in a dust-exposed occupation and during that period may have developed only a minimum case of silicosis with no or virtually no disability or lessened capacity for work. He may then have left the mining industry and worked elsewhere for a quarter of a century. If he develops tuberculosilicosis he becomes a direct responsibility of the industry for cost of medical aid, hospitalization and loss of earnings. By comparison, a person employed in any other industry or occupation who develops tuberculosis and is not subject to compensation becomes entitled to medical aid, hospitalization and social assistance if he is otherwise not able to pay. The government assumes its responsibility for the treatment, care and financial support of tuberculosis victims, provided they are not dust-exposed miners.

We suggest that the problem of the tuberculosilicotic miner is a problem to be shared by the industry and the government. Steps should be taken to relieve the industry of at least a part of its responsibility in this direction.

Mining companies are acutely aware of the threat to silicotic employees who are exposed to tuberculosis. Statistics confirm, however, that much work remains to be done to ensure as far as is possible that silicosis cases are not placed in contact with active tuberculosis cases. Obviously the industry is not able to control the activities of silicotics who have left its employ, but some system of control and checking might be possible in all cases where application for a pension has been made. It seems equally or even more important that all persons resident in mining areas should submit at regular intervals to chest x-ray and, subject to medical opinion, that tuberculin testing of all mine employees should be initiated.

The Problem of the Migratory Miner

For a variety of reasons, workmen in the mining industry move from one area to another more frequently than is the case with most other occupations. Many examples come to mind. Large numbers of miners and mine workmen were brought to Canada from Europe about 10 years ago. There were large movements of mineworkers from eastern Canada to the Alcan projects at Kitimat and Kemano. Miners from all parts of Canada moved to the uranium fields at Beaverlodge and in greater numbers to the Blind River-Elliott Lake operations. A substantial movement to Manitoba may be expected with the opening of the Thompson and Chisel Lake properties. It has been suggested, and we believe with merit, that these continuing shifts of mining population make silicosis a national problem rather than a local or provincial problem. The only organization of which we are aware that gives consideration

to silicosis on a national basis is the McIntyre Research Foundation, which by its nature is not designed to give full attention to many of the legislative and administrative problems of silicosis.

In recent years some efforts, largely unsuccessful, have been made to initiate discussions of silicosis problems on a national, or at least an interprovincial, basis. We believe this approach offers the only realistic means of confronting and eventually solving some of our problems. The following observations are indicative of the thinking of our Association on this matter.

There should be a much greater similarity of legislation throughout Canada with respect to silicosis. The present Workmen's Compensation Acts in the respective provinces now have material differences. If otherwise qualified, a miner who has moved from one province to another should not lose any part of his entitlement to compensation for silicosis. At present the provisions for qualification differ widely. The period of residence required by the different statutes varies from no specific period to five years. In order to qualify, the workman must be exposed to dust containing silica for a period aggregating two years preceding his disablement in some provinces and up to five years in others. There is a time limitation within which an application must be made which varies in the different provinces, and in at least one there is discretion in the Board to extend the time for application for an unlimited period and this discretion is widely used. Similarity of legislation would not only be of value to the workman, but would be of value to the employees and all concerned interprovincially. The same applies to rules, regulations and requirements.

Since a miner may have exposure in several provinces, or may have had exposure in other countries prior to entry to Canada, there should be similarity or requirements in all of the provinces as to pre-employment medical examination and annual examination. Such pre-employment and annual examination should include x-ray and should result in issue or refusal of a certificate of fitness for underground or other work where there may be exposure to dust. A central registry could conceivably be established for maintaining records and distributing information as to the status of miners moving from one province to another.

There should be similar and strongly enforced regulations and inspection of dust conditions, and permissible dust counts should be consistent even though the actual dust count is not necessarily the aim.

In some provinces, an award for silicosis will be prorated on the length of exposure of the workman in that province in relation to the length of his exposure elsewhere. It can and does happen that a workman otherwise entitled to a 100% disability pension may receive a meager sum insufficient for the care of himself and his family. This is not only unfair to the workman, but leads to dissatisfaction in the minds of the general public who do not understand the principles of Workmen's Compensation. On the other hand, in those provinces where the full amount, irrespective of where the exposure may have taken place, is paid, the mining industry in such provinces is paying more than should be required. The employers there are unfairly burdened with a risk and with loss contrary to the principles of Workmen's Compensation.

It is suggested that interprovincial consideration should be given to the following:

(a) That provinces prorate the cost of silicosis claims in accordance with the exposure of the silicotic in each province. This step, of course, would require establishment of similar qualifications for pensions.

(b) That the province where the claimant was last employed should accept a valid claim and seek reimbursement from the other compensation boards concerned.

(c) That the time limit for making claims be abandoned since it is long since established that the effect of silicosis is generally long delayed (i.e., after 15 to 20 years exposure). As a qualification to this suggestion, adequate employment records would be required.

(d) That the qualifying employment period in all provinces should be made uniform (perhaps two years), if indeed a time limit is essential except for those who have been exposed to dust outside of Canada.

There are, of course, many other serious problems pertaining to silicosis and its compensation. I would suggest, however, that the problems of the migratory miner, pointing up as they do the urgent need for interprovincial cooperation; the problems arising from the social service aspects of silicosis compensation, and the serious financial problems attendant on compensation costs are of a more than ordinarily urgent nature.

We, in British Columbia, do not claim to have very many of the answers. In fact we tend to look for information and advice to the larger mining provinces and Ontario in particular. We are willing to extend our fullest cooperation to any and all efforts that may overcome these problems, which we believe can be solved through the continuing efforts of all agencies and individuals concerned.

RECENT TRENDS IN SILICOSIS

Among Miners in Ontario

• W. C. WHEELER, B.A.
Chief Statistician

• A. H. SELLERS, M.D., D.P.H.
Consultant Medical Statistician

The Workman's Compensation Board of Ontario
Toronto, Ontario

Silicosis was designated as an industrial disease for compensation purposes in the Province of Ontario in 1926 and in 1928 legislative provision was made under the Mining Act of Ontario requiring that every person employed in a dust exposure occupation in a mine must have a pre-employment physical examination, including a chest x-ray, by a medical examiner appointed for the purpose. From 1925 on, a continuing program of dust control has been undertaken by the mines in Ontario.

The findings of the annual chest x-ray examinations for underground miners by physicians employed by The Workmen's Compensation Board, commenced in Ontario in 1928, were recorded on forms which were retained by the Board's silicosis medical examiners at the Examining Stations. Since 1939 surface workers have also been fairly consistently examined yearly.

Recognizing the need for statistical data on the incidence, prevalence and progression of silicosis, in 1940 the Board instituted a system for the routine reporting of all chest x-ray findings. World War II interrupted plans for the analysis of these reports, but the system continued in effect.

In 1949 a Committee, appointed by the Prime Minister for Ontario, recommended that a statistical study be made of the reports on file at The Workmen's Compensation Board and a system devised to permit follow-up of all miners until they leave the mining industry in Ontario and of all known silicotics and all those compensated for silicosis or for whom claims have been established until they die.

To implement these recommendations, a Medical Statistics Unit was set up under The Workmen's Compensation Board in 1950 to prepare and analyze data on the incidence, prevalence and progression of silicosis in the mining industry in Ontario and to study the relationship between age, length of exposure and other factors. A recording and statistical plan was drafted and approved and attention then directed toward putting the plan into complete and effective operation.

Value of the Present System

The recording and reporting system now established is not designed nor is it being used solely for statistical purposes, though perhaps the maintenance of a current picture of trends for study is still its main objective.

The recording system is being used more and more as a valuable reference source by the Chief, Chest Services Department of The Workmen's Compensation Board and by the Silicosis Medical Examiners. It would be a valuable aid to a Pneumoconiosis Research Unit if such were to be established in Ontario.

It is of interest to note that during 1959, a total of 43,558 examinations were conducted at the Miner's Chest Examining Centres operated by the Board.

Objective

The continuing objective of this study is to provide data on the incidence, prevalence, progression and mortality from silicosis among miners in Ontario. The relatively long period of exposure required for the development of this disease, the low incidence rate, and the significant movement and turnover of mining personnel, are problems which have been encountered in developing such data.

This statistical study has been conducted largely in retrospect, a fact which has added materially to the difficulties which we have encountered in attempting to reconstruct the natural history of silicosis in Ontario mines. The accumulated records relate to a large number of cohorts of miners, who have entered employment at varying times over the past 30 years or more. Many of these men change their occupation, move from one mine or mining camp to another, or leave the industry entirely, after varying periods of time. The interpretation of

any figures on the trend in incidence of silicosis is rendered most difficult by this turnover of mining personnel and effective assessment of the comparative risk of developing silicosis in various mining occupations is likewise difficult, if not impossible.

Chest X-Ray Findings

A nine-point numerical classification scheme is used by the medical examiners in reporting chest x-ray findings. The code numbers with which we are mainly concerned are "4," "5" and "6" to "9." In this presentation, however, the data relate largely to "4's" — "generalized arborization," and to "5's" — "generalized arborization with partial mottling," the first definite sign of silicosis. The special significance of new "5" ratings as our closest approach to the incidence of silicosis, is the reason why the data to be presented are confined largely thereto.

There undoubtedly have been changes in the quality and in the interpretation of the x-rays taken over the years. While this has not warranted re-reading of all accumulated films, unaccountable variations in any of the significant ratings have been made the subject of review by a member of the Silicosis Referee Board and The Workmen's Compensation Board. This has required the review of thousands of films for men with ratings in the range "4" to "9."

The Program

Having tested the method over an extended period, the following plan has been pursued:

(a) Master cards have been completed for all men with a chest x-ray category "4" to "9" at any time, commencing with the first year for which examinations were conducted and the findings recorded, and these master cards are being maintained up-to-date each year.

(b) The necessary data for all these men have been coded and punched into I.B.M. cards and appropriate tabulations prepared. Further tabulations can be made as may be indicated, either from the punched cards or from the master cards as desired.

During the past two years, attention has been turned to four special tasks arising out of suggestions or proposals made on the basis of the work done and the data produced and presented up to the end of 1956. These have been as follows:

(a) The investigation and review of all reports relating to cases rated "5" in which the recorded length of dust exposure was less than five years.

(b) The integration of medical examination reports and findings of the silicosis medical examiners with those of the Silicosis Referee Board, heretofore maintained quite separately from each other.

(c) The development of some firm data on the proportion of miners examined who have five, 10, 15 or more years exposure over the years from the date when records are first available (1928).

(d) The review of all cases in which ratings had "jumped," e.g., from a "3" to "5" or "4" to "6," and the assignment of "4" and "5" ratings to the year in which they were most probably reached.

Mine Employment Figures

In order to have some knowledge of the trend in the numbers of men employed both at risk and not at risk, available employment figures have been tabulated for each year from 1929 to 1958, for surface, underground, mill, rockhouse and other categories for each mine in Ontario. From these data, the numbers of men at risk were calculated and these figures have been used in the computation of crude rates. During 1958, the mine employment figure totalled 36,648, of which 55.4% or 20,229 were at risk (i.e., were employed in a "dust exposure occupation").

Findings

The statistical data presented relate to men from all mining camps who have been reported either by the Board's Silicosis Medical Examiners or by the Silicosis Referee Board as having a new "5" chest rating at any time from the date the examination system started (1927-28) to the end of December, 1958. Rejected applicants are excluded. Statistics to be presented relate to all men and to men with Ontario exposure only, with particular emphasis on the latter group.

In each instance the figures relate to the year when a "5" rating was assigned either by the silicosis medical examiner or the Silicosis Referee Board; they have no necessary relation to claims for compensation, to recognition thereof by the Silicosis Referee Board, or to allowance of a claim by The Workmen's Compensation Board. The figures include all known cases of silicosis to the end of 1958 excluding such cases found on original examinations (1928-30). Although the figures are firm, there may be minor adjustments.

New "5" Ratings

The number of new "5" ratings among all men examined and among men who had been employed since their last examination, by five-year periods, are shown in Figure 1.

In this and later illustrations the number of "5" ratings exclude such ratings found on original examination for employment or in em-

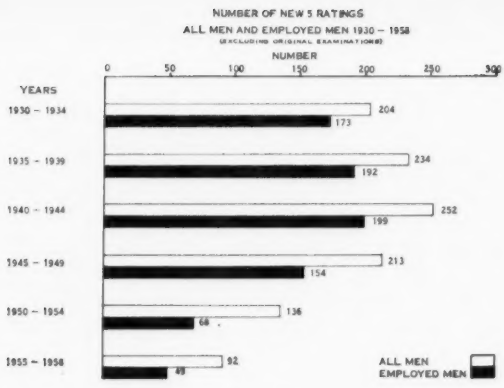


Figure 1

ployed miners when first examined when the chest x-ray program started. In excluding those men with silicosis or a "5" rating on original examination, we have excluded men who entered mining years before the medical examination and chest x-ray program went into effect and who, if x-rayed, would probably have had "5" ratings several years before, and included those miners without silicosis at that time but who later were rated "5," i.e. those men in whom the length of exposure to a "5" rating was greater.

The number of new "5" ratings both for all men and for employed men gradually increased until the period of 1940 to 1944; there was a decided decline in 1945 to 1949 and a steep reduction in 1950 to 1954. The tentative figures for 1959 would put the two values for the period 1955-1959 at 123 and 75.

Exposure Outside Ontario

The numbers of new "5" ratings among men with Ontario exposure only and among men with exposure outside Ontario are presented here in Table I.

The substantial numbers of new "5" ratings among men with exposure outside Ontario, and the increasing proportion which these cases form of the total numbers in recent years, is evident. This fact makes it desirable to attempt the computation of rates of incidence specific for men with and without exposure outside Ontario. This will be possible within the limits imposed by the recorded information, as soon as tabulations of men with five years or more exposure can be completed.

Trend in Incidence

Incidence, as distinct from prevalence, means the number of new cases of any condition arising in a given population during a specified interval of time.

TABLE I.
NEW 5 RATINGS BY PERIOD SO RATED*
(Excluding Original Examinations)

Period Rated	Ontario Exposure Only	Exposure Outside Ontario	Total
Before 1930	17	8	25
1930-34	117	87	204
1935-39	142	92	234
1940-44	158	94	252
1945-49	142	71	213
1950-54	78	58	136
1955-58	47	45	92
Total	701	455	1,156

*Regardless of employment since last examination.

The numbers of new "5" ratings recorded* for all men and for men with Ontario exposure only over the years 1930 to 1958 inclusive are presented in Figure 2. The number of employees in dust exposure (in thousands) is included for comparison.

These figures depict the incidence of silicosis among miners in Ontario as we now know it. A peak in 1941 and an encouraging but irregular decline over the years is evident. Some indication of a slight (though proportionately great) upturn seems to be suggested in 1953 and 1954 but new cases subsequently declined from 1955-1957. An upturn again is suggested by a greater number of new "5" ratings (all cases) in 1958 (22 cases) and an even greater number of "5" ratings in 1959 (31 cases). This latter figure is tentative and will shortly be subjected to review.

The number of miners at risk in dust exposure occupations has fluctuated considerably over the years, rising from a low point in the depression period to a peak of about 18,000 in 1941. In the years since 1950, the exposed population has

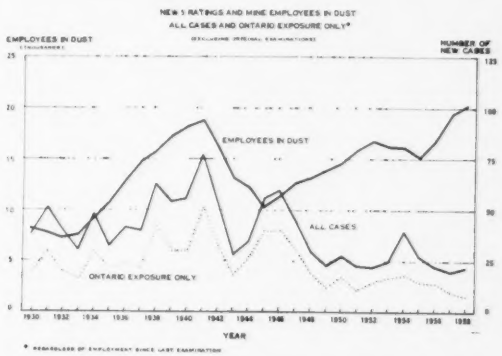


Figure 2

*Men who were never reported as a "5" but who advanced from a "3" to a "6" or from a "4" to a "6" or higher rating in successive reports have been the subject of review and estimates of when these cases first became "5" have been made by medical specialists of the Silicosis Referee Board and The Workmen's Compensation Board.

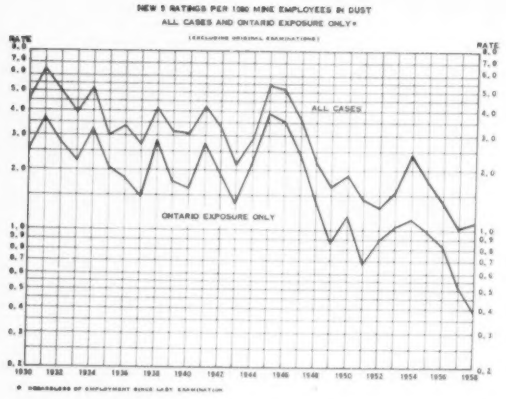

NEW 5 RATINGS PER 1000 MINE EMPLOYEES IN DUST
ALL CASES AND ONTARIO EXPOSURE ONLY*
(EXCLUDING ORIGINAL EXAMINATIONS)
RATE
ALL CASES
ONTARIO EXPOSURE ONLY
1930 1932 1934 1936 1938 1940 1942 1944 1946 1948 1950 1952 1954 1956 1958

Figure 3

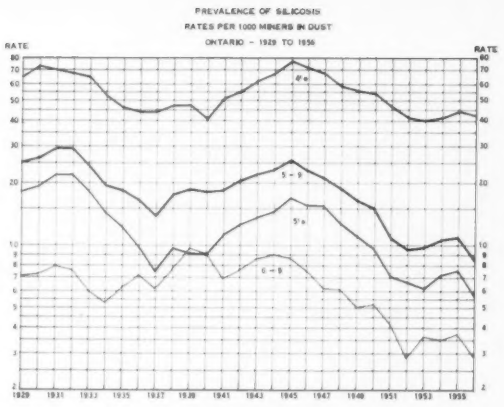


Figure 4

remained fairly constant at about 16,000 to 1955 and since than has gradually increased to over 20,000 in 1958.

The trend in the occurrence of new "5" ratings since 1930 for all cases and for cases with Ontario exposure only expressed per 1,000 employees in dust exposure is presented in Figure 3.

These rates are not true incidence rates since the composition of the risk population over the years has changed in terms of length of employment and intensity of exposure. They do, however, provide a useful measure of the problem in a relative sense at any one time.

These crude rates for new "5" ratings have declined for all cases from about five per 1,000 men in dust in 1930-34 to 3.3, 3.2, 3.5, 1.7 in succeeding five-year periods with 1.3 for the four-year period 1955-58. For men with Ontario exposure only, the rates have also declined from 2.9 per 1,000 men in dust in 1930-34 to 2.0, 2.0, 2.3, 1.0 in succeeding five-year periods with 0.7 for the four-year period 1955-58.

Prevalence of Silicosis

The complete picture of the prevalence of silicosis is not yet available. Available data do, however, give a useful indication of trends; these data are based on reports supplied by the Board's Medical Examiners over the years; they exclude cases seen only by the Silicosis Referee Board. They are simply counts of men with ratings "4," "5," and "6"-"9," for each year from 1927 on.

The complete picture can never be put together; men work in the mines for varying periods and change their occupations and are lost sight of. It is, however, likely that eventual figures will omit few cases of disabling silicosis. The available figures on prevalence, expressed per 1,000 employees in dust exposure, are illustrated in Figure 4.

Mine employment figures declined by 35% from 1941 to 1946 and only since 1956 have they risen above the 1941 figure. Most of these new employees have had little or no previous mining experience so that the rates presented in Figure 4 are over-estimates of the true trend in prevalence since World War II.

According to our tentative figures, the numbers of "4," "5" and "5"-"9" ratings reached a peak in 1941 ("6"-"9" ratings in 1939). The rates, unlike the actual numbers, show a high point in 1945 ("6"-"9" ratings in 1944) but there is another feature — a high area for all these ratings in 1930-32. From about 1941 and certainly since 1947, the trend in apparent prevalence has been downward with some flattening of trend, in numbers and in rates, since 1953.

Year of First Ontario Exposure

The distribution of the 1,156 new "5" ratings recorded to the end of 1958, by five-year periods in which the first Ontario dust exposure took place, are presented in Table II.

Of the 1,156 new "5" ratings, 1,034 or 89%

TABLE II.
NEW 5 RATINGS BY PERIOD OF FIRST ONTARIO EXPOSURE*
(Excluding Original Examinations)

Period of First Ontario Exposure	Ontario Exposure Only	Exposure Outside Ontario	Total
Before 1910	73	48	121
1910-14	124	61	185
1915-19	115	52	167
1920-24	199	100	299
1925-29	140	122	262
1930-34	36	39	75
1935-39	12	18	30
1940-44	2	5	7
1945-49	—	2	2
1950-54	—	2	2
1955-58	—	1	1
Others	—	5#	5
Total	701	455	1,156

*Regardless of employment since last examination.
#Men with no exposure in Ontario.

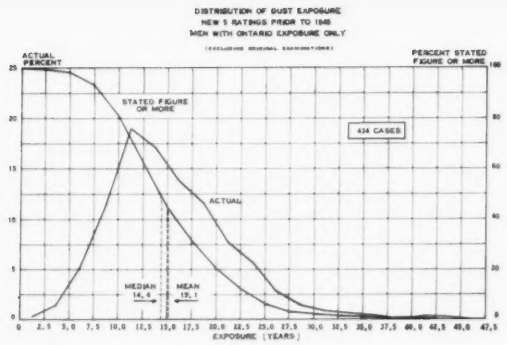


Figure 5

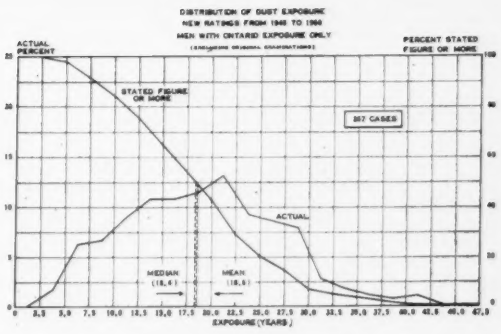


Figure 6

entered mining in Ontario before 1930; in all but 12 cases exposure began before 1940. This simply reflects the long period of exposure known to be required for the development of silicosis.

Of the 701 men with Ontario exposure only, all but 50 commenced mining in Ontario before 1930; in all but two exposure began before 1940. There have been no cases reported among men with Ontario exposure only who began mining in Ontario since 1944.

It is of interest, however, that there have been five new "5" ratings since 1944 among men with exposure outside Ontario. Two of these had exposure in Manitoba, one in Germany and two in Belgian coal mines. Tentative figures* for 1959 yield four additional new "5's" among men who first entered exposure in Ontario since 1944 but who had previous exposure elsewhere; in this case all four had previous exposure in Belgian coal mines.

Variation in Exposure

There are wide variations in the exposure apparently required by different individuals for the development of chest findings signifying silicosis, i.e. a "5" rating. To some degree, differences in critical intensity of exposure may be a factor in this phenomenon, in addition to purely individual factors.

The percentage distribution of the dust exposure times in two and one-half year intervals for 434 men with Ontario exposure only who, excluding original examinations, were first reported as having a "5" chest rating prior to 1945, is presented in Figure 5.

The median of this distribution is 14.4 years and the arithmetic mean or average is 15.1 years. In the chart is a line depicting the percentage of all the cases with a given length of dust exposure or greater, for example, 87 or 20.1% had a recorded dust exposure of 20 years or more.

*Subject to revision on review.

The comparable distribution for the 267 men with Ontario exposure only who first were reported as having a "5" rating in the years 1945-58 is shown in Figure 6.

The median of this distribution is 18.4 years and the arithmetic average slightly higher at 18.6 years. Both these values are approximately four years higher than for the previous distribution.

These data point up the wide variations in recorded exposure required by different individuals for the development of silicosis. Twelve cases of the 701 had less than five years recorded exposure; these include three men who worked in the mill for 21 months, 180 months and 192 months. In three other cases, surface employment is recorded as 84 months (mine dry), 192 months (steel shop), and 36 months surface (unspecified). Ten of the 12 cases began mining in Ontario prior to 1930; two cases began mining in 1937.

New "5" Ratings by Age — Time Intervals

Data on the mean exposure time and mean elapsed time from first dust exposure to a "5" rating and the exposure and elapsed time from a "4" to a "5," by age-group when first rated a "5," are given in Table III. The figures include all cases regardless of employment S.L.E.

The data show an increase with age in the

TABLE III.
NEW 5 RATINGS BY AGE — MEAN EXPOSURE AND ELAPSED TIME (MONTHS)*
(Excluding Original Examinations)

Age Group	No. of Cases	Mean Exposure to 5	Mean Elapsed to 5	Mean Exposure Time 4 to 5	Mean Elapsed Time 4 to 5
20-29	13	83	107	11	25
30-39	200	145	181	22	33
40-49	472	197	258	28	48
50-59	342	243	340	39	77
60 and Over	129	306	446	54	113
Total	1,156	213	288	34	62

*Regardless of employment since last examination.

TABLE IV.
NEW 5 RATINGS BY PERIOD RATED A 5 — MEAN EXPOSURE AND ELAPSED TIME (MONTHS)
ALL MEN WITH ONTARIO EXPOSURE ONLY
(Excluding Original Examinations)

Period	No. of Cases	Mean Exposure Time	Mean Elapsed Time	Mean Exposure 4 to 5	Mean Elapsed Time 4 to 5
1927-29	17	147	201	—	—
1930-34	117	173	209	16	25
1935-39	142	181	224	26	39
1940-44	158	192	253	30	51
1945-49	142	220	300	35	65
1950-54	78	218	369	39	96
1955-58	47	243	363	42	113
Total	701	197	269	32	61

mean exposure and mean elapsed times from first dust to a "5," and in the mean exposure and mean elapsed times from a "4" to a "5" rating. This finding may be associated with differences in the relative intensity of exposure or individual susceptibility, or to other factors. Of a given cohort, it is obvious of course, that those who develop silicosis at a younger age and/or after shorter durations of exposure include the more susceptible of that group as well as those with the more crucial intensities of exposure.

For the 853 men employed S.L.E., the mean exposure time to a "5" and the exposure time from a "4" to a "5" are consistently higher and the elapsed time intervals lower. This confirms the fact that men continue to advance in rating after being removed from dust exposure and suggests that there is some benefit in leaving dust exposure at a given time and after a certain amount of exposure has been obtained and that by so doing advancement to a "5" rating may be retarded.

Mean Time Intervals by Period

Data on the new "5" ratings by period when rated a "5" and the mean durations of exposure and elapsed times to a "5" rating and the intervals from a "4" to a "5" for men with Ontario exposure only, as presented in Table IV.

The average (mean) duration of exposure for new "5" ratings has increased over the years from 12 years to about 20 years. The elapsed times from first dust exposure to a "5" have increased even more sharply.

There is special interest in what has happened, over the years, to the intervals of time — both exposure and elapsed — between the first "4" rating and the first "5" rating. The mean exposure times from a "4" to a "5" rating for 529 men for whom this information is available have shown a persistent increase over the years from 16 months in 1930-34 to three and one-half years in 1955-58. The mean elapsed times from a "4" to a "5" rating for 528 men have increased from two years to nine and one-half years in 1955-58; a fact which is suggestive of changes of job in the interval.

For the 509 men who were employed S.L.E., the findings are essentially the same.

TABLE V.
NEW 5 RATINGS BY AGE AT FIRST ONTARIO EXPOSURE
MEAN EXPOSURE AND ELAPSED TIMES (MONTHS)
ALL MEN WITH ONTARIO EXPOSURE ONLY
(Excluding Original Examinations)

Age At First Ontario Exposure	No. of Cases	Mean Exposure Time	Mean Elapsed Time	Mean Exposure 4 to 5	Mean Elapsed Time 4 to 5
Under 20	117	227	323	30	57
20-29	437	197	268	31	61
30-39	119	178	233	35	68
40-49	25	171	208	28	51
50 and Over	3	129	130	24	24
Total	701	197	269	32	61

TABLE VI.
NEW 5 RATINGS BY PERIOD OF FIRST ONTARIO EXPOSURE — MEAN EXPOSURE AND ELAPSED TIMES (MONTHS)
ALL MEN WITH ONTARIO EXPOSURE ONLY
(Excluding Original Examinations)

Period Of First Ontario Exposure	No. of Cases	Mean Exposure Time	Mean Elapsed Time	Mean Exposure 4 to 5	Mean Elapsed Time 4 to 5
Before 1910	73	292	398	37	70
1910-19	239	208	295	35	71
1920-29	339	179	234	29	56
1930-39	48	139	194	31	60
1940-49	2	91	123	29	60
1950-58	—	—	—	—	—
Total	701	197	269	32	61

Age at First Ontario Exposure

The mean exposure and elapsed times to a "5" and from a "4" to "5" rating by age at first Ontario exposure, for all men with Ontario exposure only, are set out in Table V.

Here there seems to be a progressive decrease with age at first Ontario exposure in the mean exposure and elapsed times to a "5" rating but a rather curious pattern for the intervals "4" to "5." What does this mean? For men entering exposure at 30 years of age or over, only those men with greater "susceptibility" (requiring less rather than more than average exposure to develop silicosis), or with shorter but more significant exposure, would appear in our observed groups of men rated a "5" — so the findings fit expectation rather well.

For the 509 men with Ontario exposure only, employed S.L.E. when rated a "5," the intervals follow the same pattern as for all men, the averages being slightly higher for the exposure intervals and lower for the elapsed intervals as one might expect.

Year of First Ontario Exposure — Time Intervals

The mean exposure and elapsed times to a "5" rating and the mean intervals from a "4" to a "5" rating by five-year period of first Ontario exposure, for the 701 men with Ontario exposure only, are set out in Table VI.

The mean values in Table VI show a persistent decrease with period of first Ontario exposure. The long mean exposure times to a "5" relate to men entering mining in the earlier years. For men with Ontario exposure only, employed S.L.E. when first rated a "5," the findings are similar. At first sight this seems to be a disturbing contradiction but, as observed earlier, the cases entering mining in the earlier periods, tend by the nature of this analysis, to include more of the "less susceptible" men, while the cases entering mining at a later time (1930 on) and developing a "5" chest rating, tend to be "more susceptible" men of that cohort.

Year of Rating vs Year of First Dust Exposure

The distribution of new "5" ratings for the 701 men with Ontario exposure only by five-year period when so rated and by five-year period of first Ontario dust exposure, is set out in Table VII.

All the 47 new "5" ratings in 1955-58, among men with Ontario exposure only, entered dust exposure prior to 1940; four had their first dust exposure in 1935-39, seven in 1930-34 and 36 prior to 1930. No new "5" ratings were reported in this period for men who have had their first Ontario exposure since 1940. Only two cases have been reported in any period for men first exposed in Ontario after 1940 and they occurred in 1950-54.

Although the mean exposure time has increased from 12 to about 20 years, these facts are suggestive evidence of a lower incidence but not of the effective prevention and control of silicosis.

Among the 509 employed men with Ontario exposure only, there were 26 new "5" ratings reported in 1955-58; two of these had their first exposure in 1935-39, four in 1930-34 and 20 prior to 1930. No new "5" ratings have been reported to 1958 for men who have had their first exposure since 1940.

This is a most interesting finding since one might expect from past experience that at least some of the more susceptible men who began mining after 1940 would be rated "5" by 1958 since these men could have up to 19 years exposure by this time. A check of the tentative new "5" ratings reported in 1959 also reveals no new case for men first exposed in Ontario after 1940.

This could be due to many things, perhaps the most important being the pre-employment medical examination program since 1928, closer screening of all applicants since 1940, higher standards of x-ray techniques and the rejection for employment of those thought to be poor mining risks. Undoubtedly, better working conditions and more effective dust control have played a part. The role of aluminum powder administration must also be considered.

It is possible that better selection of mine personnel, dust control and other factors, may already have established a trend to lower costs in the years to come, for it seems likely that when all men who entered mining in Ontario prior to 1930 who will become disabled from silicosis have indeed been compensated, the cost of new cases for men who began mining later should be substantially reduced. The continuing costs for compensated cases, on the other hand, will offset this to some extent for a period of time since compensated silicotics are living a good deal longer now than formerly.

Aluminum Powder Administration

During the four years 1955-58, 47 men with Ontario exposure only (according to our records) were rated a "5" (Table VII). All of these men began mining in Ontario prior to 1940; 36 of them began mining prior to 1930. All of these men had had substantial periods of dust exposure prior to 1944. According to our records, all but 13 of them had had some aluminum powder. Six of these have had dust exposure since 1944. Most of the remaining 34 men had had aluminum powder administered to them throughout their exposure subsequent to January 1, 1944, that is, for periods up to and over 10 years.

This means that these men, and other new "5's" in the periods 1950-54 (78) and 1945-49

TABLE VII
NEW 5 RATINGS BY PERIOD OF FIRST ONTARIO EXPOSURE
ALL MEN WITH ONTARIO EXPOSURE ONLY
(Excluding Original Examinations)

	First Ontario Exposure											
Period Rated	Before 1910	1910 to 1914	1915 to 1919	1920 to 1924	1925 to 1929	1930 to 1934	1935 to 1939	1940 to 1944	1945 to 1949	1950 to 1954	1955 to 1958	Total
Before 1930	5	7	4	1	—	—	—	—	—	—	—	17
1930-34	20	43	26	27	1	—	—	—	—	—	—	117
1935-39	18	32	26	39	30	2	—	—	—	—	—	142
1940-44	14	19	24	51	41	9	—	—	—	—	—	158
1945-49	14	10	20	47	35	11	5	—	—	—	—	142
1950-54	7	10	12	24	13	7	3	2	—	—	—	78
1955-58	—	3	3	10	20	7	4	—	—	—	—	47
Total	73	124	115	199	140	36	12	2	—	—	—	701

(142), were not prevented by aluminum powder from developing a "5" chest rating.

This does not mean that aluminum has done nothing; but it does seem to mean that aluminum powder administration does not preclude the development of silicosis in a person who has already had some (perhaps substantial) exposure.

Future Plans

The continuing object of our program in attempting to maintain a coordinated system of medical examination records is three-fold: (1) to provide current information on the trends in silicosis among miners in Ontario; (2) to conduct such analyses of available data as may be indicated from time to time; this includes progression and mortality which have yet to be explored in a comprehensive way; and (3) to facilitate clinical study and research in silicosis among miners in Ontario by providing a complete cross-index of cases arising.

The annual silicosis costs to the mining industry continue to be high; the silicosis assessment has progressively increased since 1946. In 1958 it amounted to $2,271,000. This fact warrants continuation of the work so far undertaken.

Should a Pneumoconiosis Research Unit be established in Ontario, the central recording and statistical machinery already established would prove to be invaluable.

Comment (Dr. A. H. Sellers)

The solution of the silicosis problem will not be established, though it may be furthered or hastened, by statistics. Statistics and statistical methods can assist in seeking the truth by providing a better description of the current problem, its inherent features and its trends, and assist clinical research workers in an appraisal of hypotheses as to etiology and control.

Mortality attributed to silicosis appears to be declining slowly but it is evident that silicosis is and will be the underlying cause of or a contributing factor in a number of deaths for many years. Indeed, it is not inconceivable that the numbers of such deaths might increase in the foreseeable future.

New cases of silicosis have continued and probably will continue to arise each year, momentarily with lesser but not insignificant frequency. Currently the numbers from 1950 are about one-half of what they were prior to that time.

The great bulk of new cases (in our case "5" ratings), among men with Ontario exposure only, have occurred among men who began mining prior to 1930 — actually 651 out of 701 cases or 93%.

All but five of the new "5" ratings to date, according to our records, entered dust exposure in Ontario prior to 1945; these five all had previous exposure outside Ontario. During 1959, four more such cases were reported among men who entered mining since 1944, but all four had previous experience outside Ontario.

The silicosis problem is not under control by any of the present measures (medical examination, dust control, aluminum powder . . .) but, despite a substantial increase in the numbers of men employed in dust exposure occupations, the numbers of new "5" ratings and new cases of silicosis do not seem to be rising. Though the current trend is not unfavorable, it is not possible to say with assurance that the incidence (numbers of cases) of silicosis will not increase in the next five to 10 years.

Acknowledgements

We wish particularly to acknowledge the help of the Board's silicosis medical examiners and Dr. J. H. L. Brennan, Head, Chest Services Department of The Workmen's Compensation Board; Dr. A. R. Riddell; the Ontario Mining Association; and Mr. R. A. Holden, Unit Head, Finance-Statistical Department of The Workmen's Compensation Board and his clerical staff.

References

1. SELLERS, A. H.: Progress in Medical Statistics on Silicosis in Ontario Mines. *Proceedings of the McIntyre Research Foundation Conference on Silicosis and Aluminum Therapy* (Ontario: Schumacher, 1951).

2. SELLERS, A. H. and WHEELER, W. C.: Further Progress in the Development of Medical Statistics on Silicosis in Ontario Mines. *Proceedings of the Sixth Conference of the McIntyre Research Foundation on Silicosis* (Toronto: 1954).

3. SELLERS, A. H. and WHEELER, W. C.: Some Findings from the Statistical Study of Silicosis in Ontario Mines Undertaken by The Workmen's Compensation Board. *Proceedings of the Eighth Conference of the McIntyre Research Foundation on Silicosis* (Toronto: 1956).

4. WHEELER, W. C. and SELLERS, A. H.: Some Observations on Silicosis Statistics in Ontario. *Proceedings of the Ninth Conference of the McIntyre Research Foundation on Silicosis and Other Industrial Pulmonary Diseases* (Toronto: 1958).

Mortality from Occupational Diseases of the Lung in Ontario

• A. H. SELLERS, M.D., D.P.H.
Director
Division of Medical Statistics
Ontario Department of Health

There are few published Canadian data on the mortality from occupational disease of the lung, and specifically the pneumoconioses and silicosis with or without mention of respiratory tuberculosis.

For the purposes of this communication, three sources of information were explored: (a) The published mortality data contained in the Annual Reports, Vital Statistics for Ontario, published by the Registrar-General for the years 1941 to 1958 inclusive. In order to secure the required facts on deaths attributed to occupational disease of the lung from 1950 it was necessary to supplement this source by reference to special photostated copies of detailed mortality classifications, provided by the Dominion Bureau of Statistics. (b) All death registrations of males in Ontario on which there was any mention of occupational disease of the lung, with or without respiratory tuberculosis, during the seven-year period 1952 to 1958. (c) The official medical certificates of death of men compensated for occupational disease of the lung, dying in Ontario during the five-year period 1954 to 1958.

The Published Mortality Statistics

The comparability of official mortality statistics over the years has been disturbed periodically by changes in the International Classification of Diseases and by changes in the procedure for the assignment or selection of a single cause for tabulation in those instances in which multiple causes are recorded by the physician on the medical certificate of death.

Prior to 1950, selection of a single cause was based on a set of arbitrary rules; from 1950, with the Sixth Revision of the International List of Diseases and Causes of Death, the selection of a single cause has been based largely on the underlying cause of death as indicated by the certifying physician on the medical certificate of death (Figure 1).

The cause of death for statistical purposes is defined as the underlying cause, i.e., disease or injury which initiated the train of events leading to death. In general, the cause of death selected for official tabulation will be recorded by the physician in Part I of the medical certificate of cause of death, usually the last item in order if there is more than one entry; causes in Part II

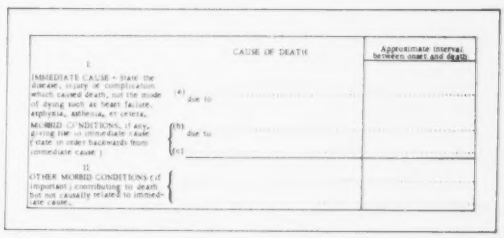

CAUSE OF DEATH
Approximate interval between onset and death
I
IMMEDIATE CAUSE - State the disease, injury or complication which caused death, not the mode of dying such as heart failure, asphyxia, asthenia, et cetera.
(a) due to
MORBID CONDITIONS, if any, giving rise to immediate cause (a) state in order backwards from immediate cause.)
(b) due to
(c)
II
OTHER MORBID CONDITIONS (of importance) contributing to death but not causally related to immediate cause.

Figure 1

being simply independent though significant contributory conditions. Thus, if a physician records "silicosis" in Part I and "hypertension" in Part II he is saying that the underlying cause of death is silicosis and the death would be so assigned.

There is no requirement, however, that a physician record all diseases or conditions present at death; if any such condition is considered by the certifying physician not to have been a significant factor in death, he may quite properly omit mention of it.

The numbers of deaths attributed to occupa-

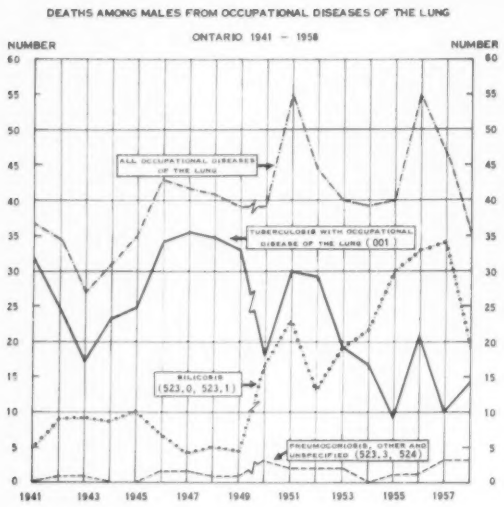


Figure 2

tional diseases of the lung* in Ontario from 1941 to 1958 inclusive, derived from the Annual Reports on Vital Statistics in Ontario, are presented in Figure 2.

The figures presented are numbers, not rates; the trends, however, would not be materially affected — perhaps accentuated — if reasonably specific rates for the group at risk were able to be computed.

The change in the cause-of-death selection procedure from 1950 appears to have had the effect of increasing the number of deaths assigned to the pneumoconioses and in particular to silicosis without mention of respiratory tuberculosis.

The total number of deaths officially assigned occupational diseases of the lung (largely silicosis), on the evidence given by the certifying physician on the medical certificate of death, has tended to increase slightly over the period but the number of deaths assigned to respiratory tuberculosis with mention of occupational disease of the lung has been gradually decreasing since 1947. This fact is emphasized by Figure 3 which gives the numbers of deaths attributed to occupational disease of the lung, with and without respiratory tuberculosis, in three-year periods from 1941 to 1958 and the percentage of the total in each period in which tuberculosis was recorded as a significant factor.

During the 18 years from 1941 to 1958, a total of 722 deaths were assigned to occupational diseases of the lung in Ontario; of this number 59.0% were due to respiratory tuberculosis with

*For the purposes of this presentation deaths from "occupational diseases of the lung" includes those which would be assigned to numbers 001, 523 and 524 of the International Classification of Diseases, 1955 Revision, W.H.O., 1957.

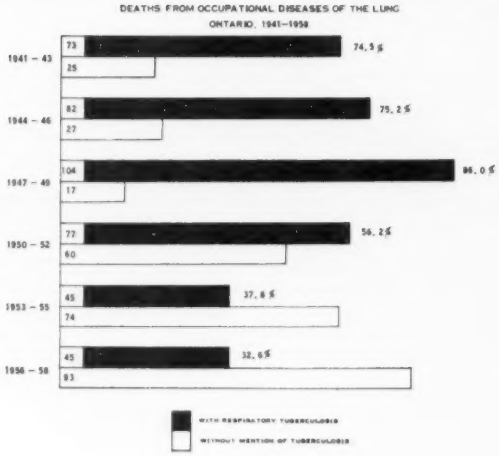


Figure 3

mention of occupational diseases of the lung. In the nine years 1941-49, this proportion was 79.0%; in the nine years 1950-58, it was only 42.4%; in the most recent three-year period it was only one-third (32.6%).

The mean (and the median) age at death — a misleading picture at best — has increased from 1941-49 to 1950-59 (Table I).

For deaths assigned to respiratory tuberculosis with mention of occupational disease of lung (I.C.D. 001) the mean ages at death were 58.1 and 64.7 years for the two periods; for pneumoconioses (silicosis) without mention of tuberculosis (I.C.D. 523 and 524), the figures were 61.3 and 65.7 years. Using the median, the figures are 59.1 and 65.8, and 61.7 and 66.9; increases of 6.7 and 5.2 years respectively within 10 years. In 1950-58, 56.3% of the deaths occurred at ages 65 and over; 12.7% occurred at ages 75 and over.

Review of Medical Certificates of Death

With a view to securing a broader picture of

TABLE I.
DEATHS FROM OCCUPATIONAL DISEASE OF LUNG BY AGE
ONTARIO — 1941 TO 1958

	1941-1949		1950-1958	
Age	001	523-4	001	523-4
Under 25	—	—	—	—
25-34	3	1	1	1
35-44	30	2	4	2
45-54	55	12	24	25
55-64	102	29	49	66
65-74	63	23	68	104
75-84	6	1	18	29
85 & Over	—	1	3	—
Total	259	69	167	227
Mean	58.1	61.3	64.7	65.7
Median	59.1	61.7	65.8	66.9

the mortality among men dying with silicosis, a special study was undertaken to provide information on the significant conditions associated with silicosis by the certifying physician on the medical certificate of death.

Through the courtesy of the Office of the Registrar-General for Ontario, transcripts of the information on all deaths in Ontario during the seven years 1952 to 1958 in which occupational disease of the lung (silicosis or other pneumoconiosis), with or without respiratory tuberculosis, was mentioned on the medical certificate, were secured for study.

There were 445 of these deaths; in 293 or 65.8% of them death was assigned to occupational disease of the lung with or without tuberculosis; in 152 deaths or 34.2%, death was attributed to another cause (Table II).

Thus, one death in every three among silicotics in this group was ascribed to a cause other than occupational disease of the lung.

TABLE II.
DEATHS WITH MENTION OF OCCUPATIONAL DISEASE OF LUNG
ONTARIO — 1952 TO 1958

Occupational Disease of Lung	Total Number	Underlying Cause	Contributory Cause
No Other Cause Mentioned	78	78	0
—With Respiratory Tuberculosis	60	60	0
—Without Tuberculosis	18	18	0
Other Causes Mentioned	367	215	152
—With Respiratory Tuberculosis	89	59	30
—Without Tuberculosis	278	156	122
Total	445	293	152
Percent	100.0	65.8	34.2

In 78 cases or 17.5% of the total, occupational disease of the lung with or without tuberculosis was the only cause mentioned on the medical certificate. In 367 cases or 82.5%, one or more other causes were mentioned.

In 119 or 40.6% of the 293 deaths which were assigned to occupational disease of the lung, death was attributed to respiratory tuberculosis with mention of occupational disease of the lung; in 169 or 57.7% death was attributed to pneumoconiosis due to silica or silicosis (occupational), and in five to other or unspecified pneumoconioses, both without mention of tuberculosis.

Respiratory tuberculosis was mentioned on a total of 149 or 33.5% of the 445 death certificates; in 89 of these, 20.0% of the total, some cause in addition to silicosis was also recorded and 30 of these latter were assigned to another cause, largely cancer and heart disease.

Silicosis or other pneumoconiosis without respiratory tuberculosis was mentioned in a total of 296 cases; in 278 of these another cause other than respiratory tuberculosis was also recorded, and in 156 of these death was attributed to occupational diseases of the lung.

The average age at death was much the same in all categories — about 66 years. It is noteworthy, however, that 38 or 13.0% of the 293 deaths assigned to occupational disease of the lung, and 20 or 13.2% of the 152 deaths not so assigned, occurred at ages 75 years or over.

All significant causes recorded on the medical certificates of death for these 445 men were coded and tabulated to permit an examination of the other conditions mentioned by the physician as contributing to death.

TABLE III.
FREQUENCY OF ASSOCIATED DIAGNOSES
445 MEDICAL CERTIFICATES WITH MENTION OF OCCUPATIONAL DISEASES OF LUNG
ONTARIO — 1952 TO 1958

Associated Condition	Times Mentioned	Underlying Cause	Contributory Cause
Asthma	7	0	7
Diabetes	4	0	4
Cancer, Respiratory System	20	20	0
Cancer, Other Sites	33	25	8
Vascular Lesions Affecting C.N.S.	23	9	14
Arteriosclerotic and Degenerative Heart Disease	137	62	75
Other Disease of Circulatory System	110	10	100
Influenza, Bronchitis, Pneumonia	79	6	73
Other Diseases of Respiratory System	50	4	46
Diseases of Digestive System	18	5	13
Diseases of Genito-Urinary System	18	6	12
Other Causes	57	5	52
Total Causes	556	152	404
Cor Pulmonale	34	1	33
Emphysema	22	2	20

In 78 deaths or 17.5% of the total, no other major diagnosis was recorded. In 221 instances one other cause was mentioned; in 110 two other causes, in 29 three, and in seven cases four other causes were mentioned.

A total of 1,001 separate disease entities were recorded on the medical certificates of these 445 men. A summary of the 556 associated diagnoses recorded on the 445 certificates in which mention was made of an occupational disease of the lung (I.C.D. 001, 523, 524), is set out in Table III.

Arteriosclerotic and degenerative heart disease was mentioned on 137 death certificates and other diseases of circulatory system in 110, together over one-half of all deaths; in 72 instances or 16.2%, death was assigned for statistical purposes to cardiovascular disease. Cancer of the respiratory system was mentioned on 20 certificates; all of these deaths were assigned thereto.

Cor pulmonale was mentioned on the medical certificate in 34 cases and emphysema in 22 cases; all but three of these deaths were assigned to other causes; 31 of the former and 17 of the latter deaths were attributed to occupational

diseases of the lung, with or without tuberculosis.

The possible association of cancer of the lung with silicosis has frequently been raised. In the review of 445 deaths during the years 1952 to 1958 in which occupational disease of the lung was mentioned as a factor in death, cancer of the lung was recorded on the medical certificate 20 times or in 4.5% of cases, four times with respiratory tuberculosis with silicosis and 16 times with silicosis uncomplicated by tuberculosis. These 20 deaths were all assigned to cancer of the lung. The number of lung cancer deaths to be expected in a group of male deaths of this age distribution is 16.3, a figure not significantly lower than the 20 deaths recorded.

The occupations recorded on the medical certificates reflected the uncertainty of routine occupational mortality statistics based on death registration data. In 91 instances (20.4% of the 445 deaths) no occupation was recorded; in 146 or 32.8% a mining occupation was given; 84 were recorded as moulders. The remainder were miscellaneous occupations in a wide variety of fields, some related and some not related to possible exposure to silica dust, many undoubtedly being temporary or short-term rather than the "usual" occupation.

Mortality Among Compensated Silicotics

Although it appears from the evidence already presented that uncomplicated silicosis may be being mentioned on the medical certificate more frequently in recent years, it is still often omitted. In many instances this omission is justified, e.g. when in the opinion of the physician the disease did not play a leading role in death, other conditions rather than the silicosis being considered to be the significant causal factor; silicotics like diabetics do die of other causes.

In order to get reasonably current information on this point, it is necessary to review the medical certificate of death of a substantial number of deaths among men with occupational disease of the lung; for this purpose, the best sample of such men would be compensated silicotics.

By special arrangement with the Chairman and the Chief Statistician of The Workmen's Compensation Board for Ontario, a list of deaths occurring among men compensated for occupational disease of the lung (largely silicotics) occurring in Ontario during the five-year period 1954-58 was secured — including name, date of birth, date of death and place of residence. There were 264 men in this group (1954 — 50, 1955 — 42, 1956 — 64, 1957 — 55, 1958 — 53), 251 of whom died in Ontario.

The necessary death searches were made by the Office of the Registrar-General for Ontario for these 251 deaths; all were promptly located and the complete medical certificate of cause of death was transcribed from the registrations to cards containing the W.C.B. information. The deaths were then coded completely as to underlying and contributory causes and the necessary tabulations made by hand. The essential findings are presented in Table IV.

TABLE IV.
CERTIFIED CAUSES OF DEATH
251 MEN COMPENSATED FOR OCCUPATIONAL DISEASE OF LUNG
ONTARIO — 1954 TO 1958

Occupational Disease of Lung	Total	Cause to Which Death Attributed				
		001	523-4	Cancer	C.V.D.	Other
With Mention of Tuberculosis (001)	55	41	—	6	7	1
Without Mention of Tuberculosis (523-4)	150	—	95	18	29	8
Total	205		136		69	
Not Mentioned	46		—	7	27	12
			136	31	63	21
Grand Total	251				115	

Of the 251 men compensated for occupational disease of the lung (silicotics) dying in Ontario during the five years 1954-58, occupational disease of lung, with or without tuberculosis was mentioned in 205 or 81.7%. In 136 or 54.2% of the total, death was attributed to occupational disease of the lung, 41 with and 95 without mention of tuberculosis. In the 69* others in which occupational disease of lung was mentioned on the medical certificate or 27.5% of the total of 251 (14 with and 55 without mention of tuberculosis) death was assigned to other causes. Of the 14 with mention of silicosis and tuberculosis that were attributed to other causes, six were assigned to cancer and seven to arteriosclerotic heart disease or cerebral embolism.

In 46 cases or 18.3% of the total, there was no mention of tuberculosis or occupational disease of lung (silicosis or other pneumoconiosis) on the medical certificate of death. In these 46 cases death was attributed to a variety of causes: cancer, seven; cerebral hemorrhage, embolism and thrombosis, seven; cardiovascular disease, 20; injuries, three; and other conditions, nine.

In 132 or 52.6% of the 251 deaths, the usual occupation was recorded on the medical certificate of death as "miner." The remaining 119 cases or 47.4% were widely distributed among other occupational categories.

*This is 33.6% of the 205 deaths in which mention of occupational disease of the lung was made on the medical certificate of death.

The mean age at death for the 264 compensated cases was 66.6 years. The expectation of life for males in Ontario at age 45 has remained at about 27.5 years from 1931 to 1956. This suggests that life may be shortened on the average by about five years as from the age at which silicosis is first recognized.

Plans for the Future

Studies now under way are designed to refine available information on mortality and to produce more precise data on the specific mortality rates and life expectancy among miners with silicosis.

To this end attention is being focused on a more effective system for the follow-up of all men with silicosis and all compensated silicotics until they die.

Summary

The silicosis "attack" rate for miners appears to be of the order of one to 1.5 per 1,000 men in dust exposure per annum; it is quite low numerically. The number of deaths attributed to silicosis each year in Ontario is small — an average of 42 per year over the past seven years.

Occupational disease of lung (silicosis) was mentioned on the medical certificate in 445 deaths over the past seven years in Ontario; in one-third of these, death was attributed to other causes.

Occupational disease of the lung is not mentioned on the medical certificate of death in about 20% of compensated silicotics dying each year; in almost half of all such cases (45.8%), death is attributed to some other cause.

In Good Taste

Those brain areas responsible for the reception and sensations of taste have been located and mapped by a University of Wisconsin scientist. Using the rat as his experimental animal, Robert M. Benjamin, M.D., of the University of Wisconsin Medical School has found that the brain's taste centers are situated in the cortex. This is something of a surprise as it has long been postulated that taste centers must be of a somewhat lesser order than the type of mental activity usually assigned to the cortex—thinking and reasoning—and that, as a result, it was believed that the taste centers would be found in the medulla or thalamus.

Benjamin also found that the taste centers are not anatomically allied to the brain's reception centers for smell. It has been long accepted that the sense of smell plays a major role in the more complex taste sensations, and it was expected that these two brain areas would be found close together.

To the contrary, Benjamin found that taste nerves from the tongue terminate in an area of the brain which serves as reception center for the nerves reporting on texture and temperature. Like the taste nerves, the texture and temperature receptors are located on the tongue and are connected with the central nervous system by two nerve cords, the corda tympanni and the ninth cranial nerve. These two nerves carry the taste, texture and temperature impulses to the medulla. From the medulla they are relayed to the thalamus, and from the thalamus to the cortex.

The portion of the brain in which the taste centers of the cortex are to be found are, in the rat, just behind the corner of each eye and beneath what might be called the temples.

By using an electrical probe which detects very slight electrical impulses, Benjamin located the areas in the brain that respond when the taste nerves of the tongue are stimulated by means of very small wires carrying an electrical charge. In effect, Benjamin set up a current along the nerve cords, and then traced the current to its terminal area in the brain.

By means of other tests, he then proved that this area was without doubt the taste center. One of these tests involved destruction of the cortical tissue in the taste area, and the demonstration that this destroyed the animal's sense of taste. Other methods of proving that this area involved taste included tests called psychological discrimination tests.

Benjamin selected rats for the initial research because they have a rather simple brain structure. Most important, the rat's brain is smooth and has no convolutions and folds which make brain-mapping more difficult in higher animals. He expects to work now, however, with cats and monkeys.

The research is part of an extensive program in the University of Wisconsin Medical School to map the functional brain centers. The knowledge will be of value in studying brain damage in humans and methods of treatment.

The Dust Control and Ventilation Services of the Quebec Metal Mines Accident Prevention Association

• GERARD GRASSMÜCK, Dipl. Ing., M.S.
Dust Control and Ventilation Engineer
Quebec Metal Mines Accident Prevention Association
Val d'Or, Quebec

The Quebec Metal Mines Accident Prevention Association was incorporated in 1940. It is managed by a Board of Directors elected at the annual general meeting of the Association's member mines. The seat of its Secretary General is at Quebec City. Its Safety Director, since 1947, is Mr. Neil H. George who is assisted by two safety engineers. A steady decline in accident frequency at all member mines has been achieved in the past 12 years: from 116 compensable accidents per 1,000 men employed in 1947 to 19.7 such accidents in 1958; a reduction of 83%.

In June, 1957, in recognition of the increasing importance of ventilation and dust control from the health, safety and efficiency points of view, the Directors of the Quebec Metal Mines Accident Prevention Association elected a committee for dust control and ventilation among its members and established a special service department, the Dust Control and Ventilation Services with headquarters at Val d'Or, Quebec.

Thus, the Services is a much younger organization than its meritorious counterpart in Ontario, the Ventilation Branch of the Mines Accident Prevention Association of Ontario, to whom we are much indebted for giving us advice in the initial stages of organizing our department.

The purpose of our new unit is to advise and assist all our members in the technical and organizational means of maintaining and improving dust control and ventilation, both underground and in surface plants. We carry out general and specific surveys and tests on ventilation and dust control systems, fans and equipment, and submit reports and recommendations. We cooperate with the mines in designing new ventilation systems and apparatus, in selecting the required equipment, and we train and assist their ventilation personnel in this field. We also maintain a collection of technical literature, of layouts and drawings and keep the mine managements informed of innovations in this branch of engineering and the prevention of pneumoconioses.

Our services are free of charge to any member mine that wishes to make use of them. We do not design dust and fume control systems in all detail but must limit ourselves to outlining and recommending the type of installation or device that we think will do the job. The design in detail must be left to the mine's engineering staff and/or to the makers of such equipment.

During these first two years these Services have concentrated on ventilation problems proper at all mines and on certain dust control problems. We will add a dust sampling and counting laboratory.

Ventilation and dust surveys have been made for years by the ventilation engineers of the Quebec Department of Mines whose painstaking and objective findings and their resulting exigencies have been of much value to us.

Our Services also submit annual reports to the Association. The reports describe activities, the state of ventilation and dust control at all member mines, and contain statistical data and a critical review with recommendations.

The following are major data from these statistics: the membership of the Quebec Metal Mines Accident Prevention Association in October, 1959, was 30 companies, but because some mines are separate units, the Services are at the disposal of a total of 37 mines, of which 13 are gold mines, 19 base and other nonferrous metal mines, and five iron mines. The territory covered is the whole Province of Quebec.

At present no mines rely solely on natural ventilation. One of the smaller mines uses a fan

in a productive portion of the mine while the inactive part is left to natural ventilation. At another smaller mine, the general ventilation system has recently been redesigned by us and should be operating shortly.

The total, installed, rated fan capacity at all mines last year was 2,270,000 cu. ft. per minute in general ventilation, and 620,000 cu. ft. per minute in auxiliary ventilation, a grand total of about 2,900,000 cfm. For 1959/60, an additional total fan capacity of 560,000 cfm has been forecast. In reality, the amount of fresh air sent underground is much higher than these roughly 3,000,000 cfm because most fans are actually operating at a lower static pressure than anticipated; therefore, they are handling more air. Also, many main fans are equipped with variable pitch blades.

We realize, of course, that it is not the rated nor even the actual volume of air handled by the fans that is a criterion of good or bad ventilation, but rather the correct distribution of the initially available air volume — in the right direction and producing adequate air velocities — and it is in this respect that much work has been done and remains to be done.

A minimum of 400 cfm of fresh air supplied to every man underground, is stipulated and supplied under favorable conditions: a larger volume under more severe circumstances. One large mine, which uses about 5,800 hp in diesel powered trackless equipment underground, multiplies the number of its men employed underground at any time by 1,500 cfm, arriving at 250,000 cfm of fresh air heated in winter and supplied by two main fans through separate intake airways.

The total footage of airways driven by all mines for this specific purpose, since the start of their operations, last year was 115,000 linear feet, or about 22 miles.

We have no producing uranium nor coal mines in Quebec, with the resulting ventilation problems inherent to such deposits. We have other problems.

(1) Sulphide dust explosions: Certain solid sulphide ore bodies are prone to cause explosions of their dusts in heavy primary or secondary blasting. Such explosions have been stopped or suppressed not only by thorough wetting but also by borrowing the principle of rock-dust dispersal from coal mining. Limestone dust is blown into the stopes, used for stemming the blast-holes, and also placed on dust barriers in adjoining mine workings, besides other precautionary measures.

(2) Sulphur dioxide: which is produced either by blasting in sulphide ores or by the spontaneous heating of old back-fill. This is combated by the systematic routing of fresh, cold air through the active workings and through the shortest possible route into the exhaust or return airways, and also by the compulsory wearing of masks, the so-called sulphur bags, by the men. These flannel bags have been treated by dipping them into a solution of soda ash and glycerine. An elaborate program of supervision and organization in case of outbreaks of fire and gases is maintained, together with refuge stations supplied with fresh fan air or compressed air at all times.

(3) Diesel-powered trackless mining equipment: This is extensively used at a single mine, Gaspé Copper. Their elaborate means for safeguarding ventilation, controlling the exhaust gases and scrubber efficiency and their fire precautions were described in detail by a member of Gaspé's staff at the Ventilation Conference of the Mines Accident Prevention Association of Ontario last year.

The regulations of the Quebec Department of Mines prescribe a minimum air volume of 75 cfm per B.H.P. of the diesel engine used in addition to normal air requirements. They also limit the carbon monoxide contents in the mine air and call for approved types of scrubbers, filters and portable fire extinguishers. At other mines, very little diesel-powered equipment is used, and if so, then under the same rigid controls.

(4) The heating and cooling of mine air and of the work environment: Heat is a major problem at only three mines. It is caused here either by the oxidation of old, pyrrhotite-bearing back-fill or by the great depth; then, special measures are taken, as at Noranda Mines which controls the air temperature by the admission of fresh air which has previously been cooled by guiding it over masses of ice built up in a large old stope during the winter, or — as at the other mines — simply by large volumes of fresh air which must be kept as dry as possible.

Thus, with these few exceptions, our mines are generally cold or cool, and wet or at least damp. Our geothermic gradient is usually high, average yearly temperatures low, and the winter long. The average surface temperatures at Val d'Or are: in summer 57.6°, winter 9.9° above zero, and only 35.3° over the whole year. Lowest winter temperatures at Val d'Or are around 40° below zero and 50° below in the Chibougamau mining region.

Seven mines have special mine air heating plants: oil burners, steam and electric coils in front of their main fans. At all other mines, it is the exchange of heat between the rock in depth and the air that warms or cools the fresh air as it is guided downwards through old workings and long fresh air ways. Ice is used inten-

tionally or unintentionally to equalize the air temperatures throughout the year.

These are our main physical problems in Quebec metal mines. Methane or hydrogen sulphide has not been observed.

Wet drilling and water blasts are standard practice, but operators must often be reminded to use sprays.

It would lead too far to mention the great number of different types of more conventional, mechanical and electrical dust and fume exhaust, air cleaning, and other devices installed at our mines, in underground crusher rooms and surface plants of all kinds. But I should mention the introduction of dust precipitation systems that use wetting agents, at one large coal handling and two ore crushing plants, by means of which the dust counts have been lowered appreciably.

The McIntyre Aluminum Powder Prophylaxis is being applied at present at 12 Quebec metal mines, seven gold mines and five base metal mines. The decision whether to introduce it rests with management. The percentage of free silica in the airborne, respirable fraction of the dust is, of course, the foremost criterion. We have had dust samples analyzed in 21 instances, by the x-ray diffraction technique, with the following results: the quartz content in the minus five micron fraction lies between 10% and 30%, average 15.3%; and for all the dust particle sizes between 9% and 43%, average 22.4%. These results do not yet cover all the mines.

After mentioning these main statistical points, a short summary on the results of our activities would be in order.

In the last two years, we have carried out at 12 mines, 12 complete ventilation surveys including ventilation plans, fan and other equipment tests, etc. Each survey is followed by a full report on the present state of ventilation and dust control and a critical review, with recommendations for alterations or improvements, if warranted.

At nine mines, this has led to the design of entirely new ventilation systems or to the redesign of existing ones, which involves, of course, the best possible routing of the air flow through the mine, with special regard to fire precautions and future mine development, mine resistance calculations, the location of fans and numerous details of ancillary installations for the proper utilization of the air and general or local dust control measures.

Ventilation systems designed by ventilation consultants are being used by six mines.

Not all mines, especially not the smaller ones, have full-time or fully trained ventilation engineers. We have, therefore, assisted or trained staff members at 21 mines in watching over their systems and installations or in seeing alterations or improvements through to completion.

Numerous specific surveys, tests, suggestions and designs in principle have been made on individual dust control problems in underground crusher rooms, smelter operations, welding and machine shops, concentrators, conveying and loading-unloading systems and operations, sampling mills and assay offices, dryers, etc.

In older crushing-grinding-screening plants where it would be even technically and operationally impossible to enclose the machinery, if we cannot enclose the dust sources, we enclose the operators, and provide them with unobjectionable air. In many such cases the men are stationary. We recommend to let these men stand or sit in booths and watch or manipulate the machinery by remote control apparatus while looking through plastic window panes. Fresh air — if necessary, heated or filtered — is forced into the cubicle by a fan from a fresh air source and distributed in the cubicle by a false pegboard ceiling or back panel. Thus, the operator is always surrounded by fresh air. Also, because the fan produces a positive pressure inside the cubicle, no dust can enter nor reach the man.

Increased attention is also being paid to small, easily mounted and low cost mobile cloth filters, such as the South African Rabson Filter, in filtering dusty air locally, for example in underground crusher rooms.

There are, of course, many new developments in the vast, highly specialized field of industrial ventilation and dust control in the whole world and we are trying to keep up with them. We feel that much more research is needed, technical or engineering research, besides and in conjunction with medical research, on the prevention of pneumoconioses. The enlightening, recent paper by Dr. John F. Paterson on Silicosis in Hardrock Miners of Ontario states that little research has been done in Canada on the different aspects of silicosis since the introduction of the aluminum powder prophylaxis in 1944. Dr. Paterson lists 32 desirable, urgent research problems and recommends the establishment of a pneumoconiosis research unit or center somewhere in Canada.

Our Services have searched the available literature for solutions to some of our technical and operational problems without finding clear, unequivocal answers that would be acceptable to our industry. As this would require original research work in well-equipped laboratories and by research staffs, we have approached the Federal and Quebec Departments of Mines and Health. To name only a few of the research problems, we were thinking of the following:

(1) The explosiveness of sulphide ore dusts in primary and secondary blasting, with special

regard to the type of deterrent rock-dust used, to prevent pneumoconioses and also undesirable effects on metal recovery because lime affects the flotation process.

(2) A critical review of all existing dust sampling and counting instruments in order to arrive at a definite conclusion on the most suitable, practical and reliable devices for routine dust sampling at our metal mines under varying conditions.

(3) The value of foaming and wetting agents in dust control and their influence on metal recovery in flotation.

(4) The mineralogical or petrographical analyses of mine dust samples.

(5) A study of the most economical and perhaps of new ways for heating the very cold fresh air supplied to mines in winter. We do not know, for example, if the "heat pump" principle could be utilized or not.

There are now at least 22 different dust sampling and counting instruments on the market in various countries. Could not there be one among them that is the answer to our prayers?

A dust sampler-counter that is being used routinely in European mines has never been tried out in Canada; it is the Leitz Tyndalloscope. This is a light, portable, direct-counting device that operates on the principle of light dispersion by dust clouds and eliminates the time-wasting counting of dust particles under the microscope. Another dust sampler-analyzer is under development which automatically and quantitatively separates and determines the total dust load in particle sizes above and below five microns, while being operated in a stream of dust-laden air. Its purpose is to collect respirable dust in a quantity sufficient for subsequent mineralogical, x-ray and chemical investigations which then permit accurate assessment of the degree of the silicosis hazard presented by a given mine dust.

As regards wetting and foaming agents to bind dust, we know pretty well the degree of their effectiveness or limitations, and so do the Europeans, who have now introduced bentonite — a clay commonly applied in oil well drilling and other fields — instead of chemicals and detergents for dust suppression, with excellent results.

I may also mention some recent entirely new principles and techniques that I have come upon in European literature and which may or may not be known here.

(1) The stemming of drill holes using plastic cartridges filled with water or weak solutions of chemicals. The purpose is to precipitate the dust and to absorb the harmful gases in blasting fumes. The technique proved to be safer than the usual ones; an economy of 10% in explosives was effected, part of the harmful gases was absorbed, and the respirable dust fraction was reduced by up to 80%.

(2) Experiments led to the discovery that the mineral constituents of mine dusts have different natural electrostatic charges or polarities. The pathogenic minerals, like quartz, appear to be predominantly negatively charged, while the inert or neutral particles carry a positive charge. The logical conclusion was to bring them together, so that they would neutralize each other's charges, agglomerate and settle out. A cloud of harmless dust is blown into dust-laden mine air. Surprisingly enough, not only is the total dust concentration reduced instead of increased, but the respirable fraction of harmful dust was reduced by about 75%. Similarly favorable results are obtained by the stemming of the drill or blast-holes using neutral rock dusts like limestone and others. The tests are being continued on a large scale. They are of particular significance to hot mines because the dust precipitating agent is a dry substance, and the humidity of the ambient is thereby not increased, as it would be by wetting.

(3) The Silicosis Research Institute of the Association of the Mining Profession, as it is called in West Germany (where there exist, incidentally, three such Research Units) establishes and assesses mathematically the degree of dangerousness — as they call it — of each mine or mineral deposit with respect to silicosis; i.e., the silicosis hazard at each mine.

The multiplication of the following values or factors leads to a final characteristic figure for each mine: (a) The petrographical or mineralogical composition of the rock or ore. Hereby an empirical factor of 1.0 is assigned to quartz, 0.7 and progressively less to feldspar, the different micas, clay, ores, coal, fluorite, etc., down to a value of 0 for calcium carbonate and lime. This is the rock or ore value or characteristic. (b) The second value is the percentage of the respirable portions of the different minerals in the dust. This is the size value or characteristic.

If one multiplies the two and totals the products, a mine dust characteristic is arrived at. It often happens that a highly siliceous mine or deposit offers no higher a silicosis hazard than a low-silica one, simply because less high-silica respirable dust is produced by the particular mining methods, rock characteristics, etc. in the high-silica mine.

If the mine dust characteristic is now multiplied by the number of years during which a miner has been exposed to the corresponding dusts, and the products are totaled, an ultimate figure is arrived at, which is applied in assess-

Example: a) Mineral composition of the respirable dust

	Factor	%	Mineral value
Quartz	1.0	23	23.0
Mica	0.5	25	12.5
Clay subst.	0.2	24	4.8
Ore	0.1	12	1.2
$CaCo_3$	0.0	16	0.0
Mineral Characteristic			41.5

b) Dust concentration $<10\mu$: 20 mg/m³

c) Calculation of the dust characteristic accdg. to the formula:

$$\text{Dust characteristic} = \frac{\text{Dust conc. } <10\mu \times \text{Mineral characteristic}}{10}$$

$$\text{Dust characteristic} = \frac{20 \times 41.5}{10} = 83$$

Dust characteristic below 50, harmless; above 100, definitely harmful.

ing the share of different mines in the compensation of a silicotic miner, in other words for prorating the total compensation payment.

In this way the West Germans have been able to classify their mineral deposits according to the degree of the silicosis hazard that each mine presents. That this has not only become an accepted practice but also that no secret is made of it, is shown by the fact that they even have an Atlas of Germany entitled *Atlas for the Characterization of the Silicosis Hazard Degree of Rocks and Economic Minerals of German Mineral Deposits.* This atlas was compiled and published by the Silicosis Research Institute at Bochum in 1947.

Such determinations require, of course, a great deal of laboratory work, reliable statistical data and a complete over-all organization. One should not jump to the conclusion that we should attack the same problems in the same grand way. Our conditions are different, and, above all, the number of our men employed in mining is much smaller and the silicosis incidence lower. But I do suggest that research is necessary and urgent and we can start on it by tapping hitherto little used sources of information: the world literature dealing with this field. If we conscientiously study such publications, carefully test and intelligently apply those ideas, principles and techniques that appear to be promising under our conditions, we shall save much time and effort and render a great service to our industries, to the men that work in them, and to our country.

The State of Surgery

Women may be the stronger sex in terms of longevity but they are still the weaker sex when it comes to surgery. The fact that women tend to have more operations than men, and take longer to recover from them, are among major findings on surgical procedures presented in the current issue of *Patterns of Disease.*

Statistics show that in short term hospitals, about five operations per 100 women are performed each year compared to four for every 100 men. If deliveries are included, the women's rate rises to 8.5 per 100.

Of the approximately 7.1 million operations performed every year in this country, the commonest procedure of all is tonsillectomy and/or adenoidectomy, while appendectomies come second. For both these operations, and also for the reduction of fractures and dislocations, the incidence rates for men and women are approximately the same. With rates on other types of operations, however, the difference between the sexes is marked. Hernia, for example, which primarily affects men, accounts for 4.6 operations per 1,000 males and only one operation per 1,000 females. In contrast to this are figures for genital procedures: 14.5 per 1,000 females and 3.1 per 1,000 males.

Who does the surgery? Although the current trend is toward specialization, the role of general practitioners in surgery cannot be overlooked. An estimated 44% of these physicians in the United States perform minor surgery.

However, the general practitioner is steadily giving way to the specialist in the American surgical scene. In the United States between 1950 and 1958, the total number of physicians increased by 9.5%; full- and part-time surgeons by 26%, and anesthesiologists by 126%; while general practitioners decreased by 6%.

Specialization influences the estimates which surgeons make about the length of a patient's convalescence. Professors and specialists make a shorter estimate of convalescence needed before a surgical patient returns to work than do general practitioners. A study conducted among physicians revealed that with appendectomies, for example, professors of surgery estimated an average of 12 days convalescence before returning to light work, board certified surgeons estimated 13 days, and general practitioners 15 days. For herniorraphies, the professor of surgery's estimate averaged 20 days, and the general practitioner's, 28.

And patients take their physicians' advice seriously. The opinion of the physician appears to be the most important single factor in determining the duration of total convalescence after uncomplicated surgery. Other factors such as age, income and sick leave benefits seem to have little or no effect on length of convalescence.

• PROFESSOR J. GOUGH
The Royal Infirmary
Welsh National School of Medicine
Cardiff, Wales

EMPHYSEMA in Relation to Occupation

In Britain emphysema is frequently associated with chronic bronchitis; so much so that when the term chronic bronchitis is used the word emphysema frequently follows, indicating the common association of the two conditions. Chronic bronchitis with emphysema is much more common in men than in women and this raises the question whether this difference is related to occupation. In order to get more precise information it is first necessary to recognize that there are different anatomical varieties of emphysema and these have different causations. To arrive at agreed terminology, definition and classification of chronic pulmonary emphysema, a Ciba Guest Symposium[3] has proposed a classification based on morbid anatomy since too little is known about the pathogenesis to justify a formal pathogenic basis. Emphysema is defined as a condition of the lung characterized by increase beyond the normal in the size of air spaces distal to the terminal bronchiole either from dilatation or from destruction of their walls. Alternatively it may be defined as abnormal dilatation of those parts of the lung that bear alveoli (Heppleston 1953, 1954). As these anatomical parts comprise what is called the 'acinus' in the German literature, the term acinus was used in the Ciba classification.

Emphysema may affect the lung as a whole or it may affect varying numbers of individual lung lobules (Gough[4]). The lobule forms an important anatomical reference for distinguishing various forms of emphysema. When the proximal elements of the acinus are involved, i.e. the respiratory bronchioles, the emphysema will be seen to affect the central parts of the lobules. When the alveolar ducts and the distal alveoli are principally involved the emphysema will be seen related to the interlobular septa. If the emphysema affects all the structures it is referred to as panacinar or as Wyatt[18] has called it, panlobular. Another distinguishing feature is whether the emphysema consists of dilatation alone or whether there is also destruction of the walls separating the air spaces. The anatomical distribution together with the presence or absence of destruction forms the basis of the Ciba classifications. Two alternatives are suggested, the ingredients are the same in both but in the one the primary distinction is the anatomical distribution, whereas in the other it is presence or absence of destruction.

1. Unselective distribution beyond the terminal bronchiole (panacinar emphysema)
   a) Dilatation alone (e.g. compensatory emphysema and emphysema due to partial main bronchus obstruction)
   (b) Destruction of the walls of air spaces (panacinar destructive emphysema)
2. Selective distribution beyond the terminal bronchiole
   (i) Predominantly affecting respiratory bronchioles
      (a) Dilatation alone (e.g., focal emphysema due to dust)
      (b) Destruction of the walls of air spaces (centrilobular emphysema)
   (ii) Predominantly affecting alveolar ducts and sacs
      (a) Dilatation alone, and (b) Destruction
3. Irregular distribution beyond the terminal bronchiole (irregular emphysema)

The following alternative arrangement of the classification was proposed:

1. Dilatation alone
   (a) Unselective distribution (compensatory emphysema and emphysema due to partial main bronchus obstruction)
   (b) Selective distribution predominantly affecting respiratory bronchioles (e.g. focal emphysema due to dust)
2. Destruction of the walls of air spaces
   (a) Unselective distribution (panacinar destructive emphysema)
   (b) Selective distribution predominantly affecting respiratory bronchioles (centrilobular emphysema)
   (c) Irregular distribution (irregular emphysema)

(Subgroups with selective distribution predominantly affecting alveolar ducts and sacs are omitted from this arrangement because those who favoured it did not recognize their existence. If later experience shows them to exist, they

can be inserted in the appropriate positions under 1(b) and 2(b).)

Technique

In the post-mortem study of the anatomy of emphysema expansion of the lungs is essential and it is now generally recommended that some form of large lung sectioning should be adopted on the expanded fixed lung. Sections may be obtained by free hand cutting and the tissue examined, preferably under water or by machine cutting, mounting the sections on paper as in the Gough and Wentworth technique. The latter preparations have the advantage of easy storage and they provide ready distinction of the more important types of emphysema. An up-to-date account of the technique is given by Gough.[5]

Serial sections studied microscopically are necessary for the identification of the particular air spaces involved. Histology is also required to determine the presence of bronchiolitis. A further technique which is of value for research purposes is that of cine-photography of consecutive serial sections (Heppleston.[10])

The Ciba Symposium also proposed standards for grading the severity of emphysema. Three grades are used, mild, moderate and severe, and in the 1959 publication photographs are given of suggested standards. In cases of focal and centrilobular emphysema the severity is assessed on the amount of respiratory tissue affected by the emphysema. In mild cases less than 25%, moderate cases 25% to 50%, in the severe cases more than 50% of the lung should be abnormal. In the panacinar emphysema size of the air spaces is the main basis of grading and comparison is made with standards.

Focal Emphysema

One form of emphysema which is certainly due to occupation is the focal emphysema due to dust. In large sections the dilated spaces can be seen in and around dust deposits towards the middle of the lobule. In the early stages the emphysema is strictly confined to the middle parts of the lobule but in the advanced stages may have extended to involve the whole of the lobule. The dilatation specifically involves the respiratory bronchioles. Heppleston[8] has shown that there is no bronchial or bronchiolar obstruction to account for the emphysema but that it is due to the consolidation of the air spaces around the respiratory bronchioles by macrophages containing dust. The shrinkage of these lesions leads to a greater inspiratory pull on the walls of the respiratory bronchioles. Atrophy of the muscle in the walls of these bronchioles also appears to be a contributory factor. Focal emphysema is seen characteristically with such dusts as coal and indeed it is one of the common lesions in the simple pneumoconiosis of coal workers. It is also seen with other dusts of low free silica content such as natural graphite but it may also occur with the purest carbon indicating that it is a process quite different from silicosis. Focal emphysema may be seen in the lungs of the general population apparently due to the polluted atmosphere, but whether it is the dust or other pollutants which causes the focal emphysema in city dwellers is not known.

Emphysema in Silicosis

Advanced cases of simple silicosis may occur without any evidence of emphysema and in comparison of a large series of cases it can be seen that emphysema around classical silicotic nodules is much less frequent than around coal nodules. Heppleston[9] considers that this is due to the fact that the silicotic nodule during its formation compresses the respiratory bronchioles which is the reverse of what happens with the coal nodules. However, emphysema may occur around silicotic nodule and this appears to be due to the extension of the silicotic process into the interalveolar septa around the nodule leading to destruction of some of the alveolar walls. In cases of advanced conglomerate silicosis or where there has been much fibrosis due to coexistence of tuberculosis, there may be severe emphysema in the nonsilicotic parts of the lungs which may be due to the so-called vicarious enlargement but more often due to associated chronic bronchitis.

Emphysema Due to Cadmium

Special reference may be made to chronic cadmium poisoning since the inhalation of cadmium oxide leads to a severe form of destructive emphysema. Such cases have been reported much more frequently in Europe than in the U.S.A. Acute cadmium poisoning due usually to a single episode of inhalation of cadmium oxide may cause death within a few days due to acute pneumonitis in which there is oedema, hyaline membrane formation and striking proliferation of the cells lining the air spaces. Chronic changes, however, due to smaller inhalation over long periods causes emphysema. Lane and Campbell[14] noted that the severe emphysema spared the periphery of the lung but on close analysis I consider that this appearance is due to the escape of the periphery of some of the individual lobules and that the condition is a severe form of centrilobular emphysema.

Centrilobular Emphysema

When studying large sections mounted on paper, I soon became familiar with the appearances

of focal emphysema due to dust. I noticed, however, that in the lungs of some miners and also in the lungs of emphysematous patients in the general population, there were instances of emphysema towards the middle of the lobules not associated with dust (Gough[4]). Histologically these showed chronic bronchiolitis. This form is now generally called centrilobular emphysema (Leopold and Gough[15]). It is of common occurrence and important in terms of disability and as a cause of death. It has been found in other parts of the world besides Wales; Australia (McLean[16]), London (Heard[7]), U.S.A. (Wyatt[18]). Many of the affected individuals also have inflammation of the large bronchi. Chronic bronchiolitis with centrilobular emphysema comprise a high proportion of the cases of nonspecific chronic respiratory disease in the general population in Britain. The comparative features of focal and centrilobular emphysema are given in the following table (Gough[6]).

Focal Emphysema	*Centrilobular Emphysema*
A uniform distribution in the lung is usual	Often patchy and may be quite localized
Nondestructive (fusiform dilatation of respiratory bronchioles)	Destructive (disintegration of distal respiratory bronchiole)
Dust sheathing of the spaces	Only normal quantities of dust
No inflammatory changes in the bronchiolar pathways	Bronchiolitis and inflammatory change within the parts of the lesion itself
Normal airway calibre	Normal, dilated or narrow airways

Whether bronchitis is caused or aggravated by occupation is as yet uncertain although intensive investigation is being carried out to determine this. Although the national statistics for England and Wales show that miners and foundry workers have an excessive mortality and morbidity from bronchitis compared with other workers, as Higgins et al.[11] point out, and although this at first sight might suggest that these occupations are important factors, it is noted that the wives of these workers also had a high incidence for bronchitis. Several research workers have concluded that miners are unduly prone to bronchitis. Bohme and Lent[1] found the incidence of bronchitis almost twice as great in miners as in nonminers. Pemberton[17] concluded that United States miners had an increased incidence of chronic bronchitis and emphysema. In British communities Carpenter et al.[2] and Higgins et al.[12] found that miners and ex-miners had more bronchitis and respiratory disability than nonminers of a comparable age. Higgins et al.[12] found that miners and foundry workers are more prone to bronchitis than other industrial workers but it is not yet certain that this excess is due to occupational factors. Keatinge et al.,[13] however, found little evidence to support the belief generally held that bronchitis is a common condition in foundry men. The question of whether occupation contributes to the incidence of chronic bronchitis can only be determined by comparative community surveys. There is nothing in the anatomy of individual cases to resolve the difficulty and centrilobular emphysema due to bronchiolitis may be difficult to distinguish from focal emphysema due to dust on naked eye examination.

There is an important relationship between the presence of bronchitis and the accumulation of dust in the lungs. The relationship is an inverse one, that is to say if there is chronic bronchitis less dust will be accumulated in the lungs. This is presumably due to the dust being swept out in the increased secretions. Even if dust is already in the interstitial tissues of the lungs there is evidence that with the subsequent development of bronchitis some of the dust may leave the lungs as the machrophages which contain it migrate in to the lumen of the bronchi under the stimulation of the inflammation of the latter. The important practical side of this is, that of men working in a dusty occupation those who become most disabled may well be those which develop chronic bronchitis but they may develop no radiological evidence of pneumoconiosis, while others with healthy bronchial trees may show little or no disability but develop radiological signs of pneumoconiosis. If, as is usually the case, the payment of compensation requires radiological evidence of dust disease, those with chronic bronchitis and normal chest radiographs may feel unjustly treated. It is obviously therefore of great importance to try and determine whether dust or other factors in industry contribute to the development or aggravation of bronchitis, so that preventive measures may be sought and that in the meanwhile the workmen be justly treated.

References

1. Bohme, A., and Lent, H.: *Beitr.Silikose Forsch. Heft* 11, p. 1 (1951).
2. Carpenter, R. G., Cochrane, A. L., Gilson, J. C., and Higgins, I. T. T.: *Brit.J.industr.Med.* 13:166, (1956).
3. Ciba Guest Symposium: *Thorax.* 14:286 (1959).
4. Gough, J.: Proc.Royal Society of Med. 45:576 (1952).
5. Gough, J.: in *Modern Trends in Pathology* (London: 1959, Butterworth & Co.)
6. Gough, J.: *Harvey Lectures 1957/58*, Series 53, 171, Acad. Press New York, 1959.
7. Heard, B. E.: *Thorax.* 13:136 (1958).
8. Heppleston, A. G.: *J. Path.Bact.* 66:235 (1953).
9. Heppleston, A. G.: *J.Path. Bact.* 67:51, (1954).
10. Heppleston, A. G.: *Lab. Invest.* 4:374 (1955).
11. Higgins, I. T. T., Oldham, P. D., Cochrane, A. L., and Gilson, J. C.: *Brit.Med.J.* 2:904 (1956).
12. Higgins, I. T. T., Cochrane, A. L., Gilson, J. C., and Wood, C. H.: *Brit.J.industr.Med.* 16:255 (1959).
13. Keatinge, G. F., Buxton, E. R., and Hunt, P. J.: *Brit.J. Dis.Chest.* 53:333 (1959).
14. Lane, Ronald E. and Campbell, A. C. P.: *Brit.J.industr. Med.* 11:118 (1954).
15. Leopold, J. G. and Gough, J.: *Thorax.* 12:219 (1957).
16. McLean, K. H.: *Aus.Ann.Med.* 5:73 (1956).
17. Pemberton, J.: *A.M.A. Arch.industr.Hlth.* 13:529 (1956).
18. Wyatt, J. P.: *Amer.Review of Respiratory Diseases* 80: 94 (1959).

ROENTGEN STUDY OF SILICOSIS

in Ceramic Workers Who Have Received McIntyre Aluminum Powder

- J. W. G. HANNON, M.D., F.C.C.P.
  Medical Director
  McIntyre Research Foundation
  Washington, Pa.
- PAUL G. BOVARD, M.D., F.A.C.R., F.I.C.R.
- LESLIE R. OSMOND, M.D., F.A.C.R.
  Consulting Roentgenologist
  Tarentum, Pa.

One of the important potential health hazards in the ceramic industry is the presence of air born silicious dusts. These dusts when inhaled and retained in the lungs in sufficient concentrations can produce silicosis, a fibrotic condition of the lungs. Silicosis occurs in many stages of severity and in severer forms is capable of causing disabling pulmonary symptoms.

This study deals with a ceramic plant that was faced with the silicosis problem. The plant manufactured ceramic sanitary bathroom fixtures. The raw materials used in the manufacture of the products were ball clays, china clays, feldspar and flint (99.2% SiO_2). These materials were received in bulk and the particle size small enough that the physical state could be compared to fine dust. The handling of the raw materials was associated with the production of air-borne dusts that carried a potentially serious hazard. This study is limited to the casting shop employees who make up most of the exposed employees.

The dust counts in the casting shop showed wide variations in the peak dust exposure during the eight hour work day but the average dust exposure was computed at 7.0 million particles per cubic foot. Air-borne dust analysis by chemical and physical methods was not performed but studies of settled dusts, analyzed by x-ray diffraction and petrographic analysis, disclosed the following on repeated examinations: quartz, 25%-30%; feldspar, 25%-45%; kaolinite, 25%-40%; opaque, 2%-3%; carbonates, trace; and rutile, trace.

It is a well known fact that settled dust is seldom of the same composition as air-borne dust, however it does give some indication of the potential aerial contaminants.

At the time of the original study (1944) soda ash was added to the casting mixture as a deflocculent in order to keep the particles in the casting slip in suspension. Soda ash[1,2] in concentrations as low as .006% markedly increases the solubility of silica and produces an active surface on the silica particle. The statistical finger of guilt has been pointed at soda ash in ceramic casting and the abrasive soap industry as the mechanism causing precocious silicosis.

Plant management was cognizant of the problem and in the 1920's applied recognized measures for dust control but without the expected success. As engineering control advanced further, measures were taken but in spite of all attempts it was found that although the hazard was mitigated, complete eradication was not achieved and new silicotics were being developed each year.

In 1944 it became apparent that dust control, medical control and educational programs were not able to eradicate the silicosis hazard that existed. The president of the firm as well as the medical director visited the Saranac Laboratories as well as the McIntyre Research Foundation before asking permission to use aluminum therapy and prophylaxis as a further aid in the solution of the problem.

The program was installed in 1944 and the objectives were to determine if silicosis could be prevented by the inhalation of McIntyre aluminum powder as had been proven conclusively in animal experiments.[1,2,3] The objectives of the program may be enumerated as follows:

1. Can the inhalation of McIntyre aluminum powder prevent silicosis in human workmen exposed to silica dust?

2. Will the inhalation of McIntyre aluminum powder prevent the usual progression of early already established silicosis?

The men chosen for the study were free from active tuberculosis, but were in many age groups with varying years of exposure in the casting department of the plant. Some had previous exposures to silica and other dusts. As this particular plant was not directly connected with defense work during the period of World War II, the preplacement physical standards were lowered through necessity.

The administration of McIntyre aluminum powder was made with a small mill which produced the powder as the men took treatment. The average treatment time was six minutes inhalation once a week for approximately 50 treatments a year. The participation in the program although not perfect was good in most instances. Some men missed few or no treatments while others took treatment on a sporadic basis during this study over a period of 15+ years. Ceramic silicosis is progressive and the workmen continued at work and exposure during the period of this study.

We were of the firm belief that the development and progression of silicosis in this plant could be measured by the use of x-ray films. The results of the initial x-ray study of the men in 1944 is shown in Table I. It can be seen that many of the workmen had long exposure to silica and had radiological evidence of silicosis. The x-ray classification used in this study is by Bovard and is as follows:

1 Perfect Chest.
1+ Increased lung markings but chest within normal limits.
2 Generalized arborization, suspicious for early silicosis.
2+ Generalized arborization with partial mottling.
3 Classical silicosis.
3+ Early coalescence or pleural reaction.
4 Massive lesions in both upper mid zones.
5 Any complications of 2 to 4.

TABLE I.
X-RAY CLASSIFICATION OF WORKERS (1944)

Years Exposure	1+	2	2+	3	3-5	4	4-5	Total
20-25		1	3	4		1	2	11
15-20		5	3	2	1		1	12
10-15		7						7
5-10	3	10	1					14
1-5	6	56						62
0								
In 1944	24	2						26
Total	33	81	7	6	1	1	3	132

Table II shows the age, exposure in years, the classification in 1944 and changes in 1959 and whether the men availed themselves of the opportunity to receive the aluminum inhalations.

It is significant that all six of the seven men who showed progression had from 10 to 25 years exposure to silica dust before the treatment was instituted at the plant, and it is our opinion based on surveys in other industries over a period of 25 years that progression is inevitable after this length of exposure even without further exposure to silica.

Of the men who were hired in the period 1940-1945 only one showed slight progression (1+ to 2) and he had four years of silica exposure prior to being hired.

We feel that another significant finding in this survey was the absence of roentgen evidence of complicating infection by tuberculosis or tumor formation in 132 men over a 15 year period.

TABLE II.

Name	Age	Years Exposed 1959	X-Ray Progression 1944	X-Ray Progression 1959	Prescribed Aluminum Inhalations Taken
B,E	60	38	4	4-5?	70%
K,C	58	32	3	4	18%
L,G	59	40	3	4	59%
L,J	51	35	2	2+	10%
S,H	56	26	2	2+	5%
T,C	55	26	2+	3	54%
D,J.W.	37	17	1+	2	15%

During the period of this study the management and union of the plant gave their utmost cooperation and it is the opinion of both groups that aluminum therapy is not a substitute for dust control but it does play a part in the prevention and progression in silicosis as a third aid to dust control and medical control. Both management and the union are of the opinion that engineering methods have done much to minimize the potential hazards of airborne silica dusts but they are also cognizant of the fact that it is impossible to control the actions of the workmen either individually or collectively at all times and in all places. It is a well known fact that dust control in some plants is dependent upon the cooperation of the workers and if such cooperation is not obtained then the dust counts can soar to great heights.

Conclusions

(1) The inhalation of McIntyre aluminum powder can help protect the worker from silicosis over 15+ period of years. (2) The use of McIntyre aluminum powder will prevent or decrease the rate of severity of the usual progression of ceramic silicosis. (3) There was no evidence that the inhalation of McIntyre aluminum powder was harmful in any manner to the workmen who took this type of additive prophylaxis in the prevention of silicosis.

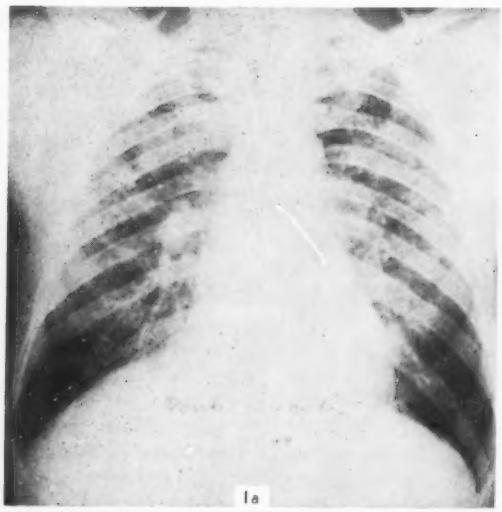
References

1. DENNY, J. J., ROBSON, W. D., and IRWIN, D. A.: Prevention of Silicosis by Metallic Aluminum. *Canadian Med. Assoc. J.*, 37:1, (1937).

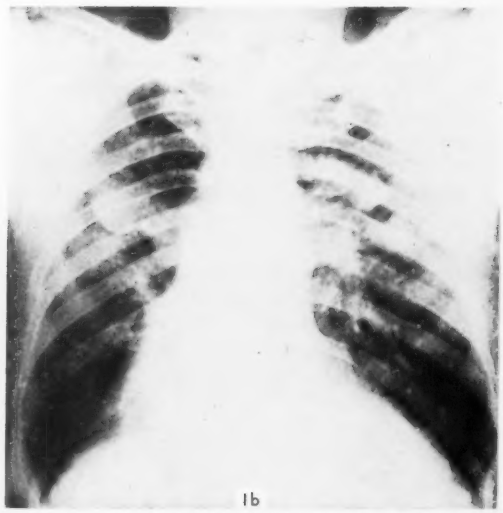
2. DENNY, J. J., ROBSON, W. D., and IRWIN, D. A.: Prevention of Silicosis by Metallic Aluminum. *Canadian Med. Assoc. J.* 40: 213, (1939).

3. GARDNER, L. U., DWORSKI, M., and DELAHANT, A. B.: Aluminum Therapy in Silicosis. *J. Ind. Hygiene and Toxicology*, Sept., 1944.

The evidence of progression as measured by x-ray films in the seven workmen in Table II can be demonstrated by the three following comparative x-ray studies:



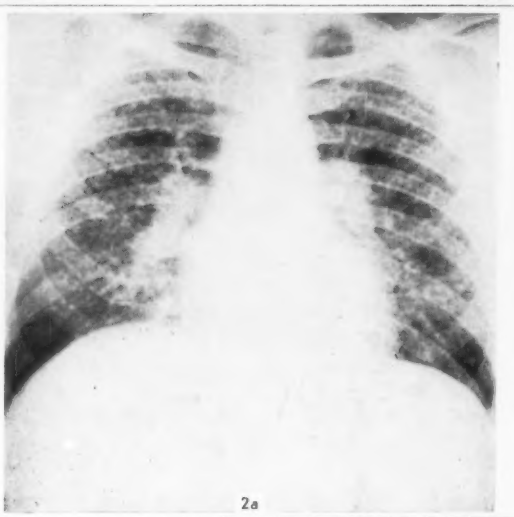
1a



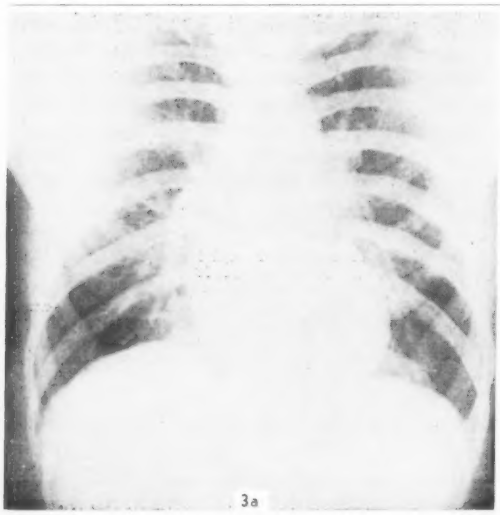
1b

Code - G,L Fig. 2, a, b, c.

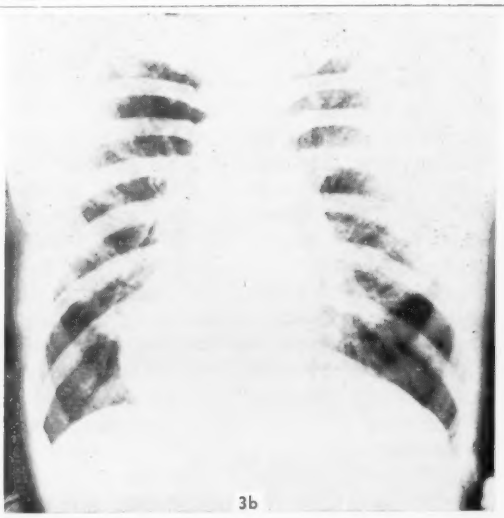
The first film in 1944, after an exposure of 24 years, shows uniform bilateral formation, without any evidence of complicating infection and no pleural reaction. Subsequent film made in 1948 shows no significant progress, but the film made 10 years later, in 1958 shows definite evidence of increased density in the periphery on both sides with a tendency toward coalescence with the nodules and no evidence of complicating infection.



2a



3a



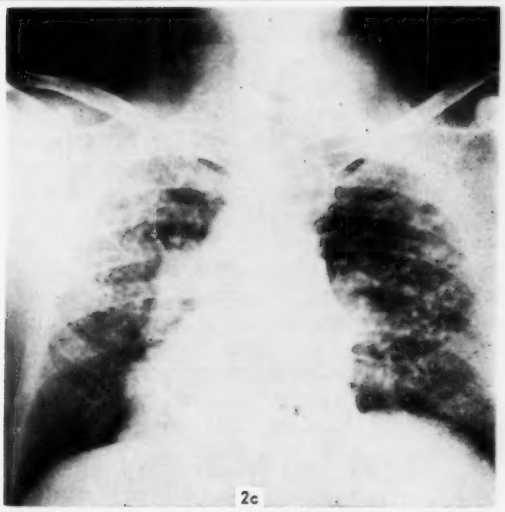
3b

1c

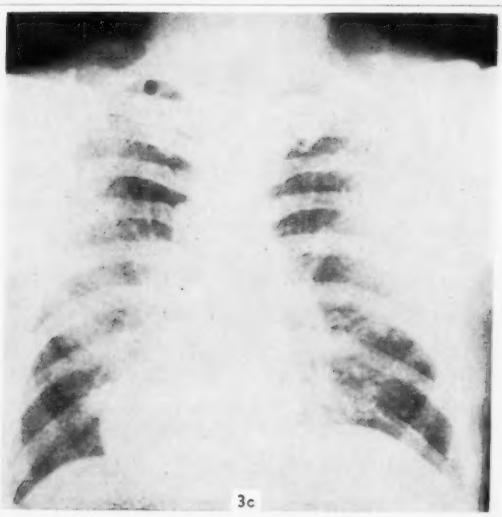
Code - B,E Fig. 1, a, b, c.

The original film made in 1944 shows early massive lesions in both upper mid zones. There is slightly more progression on the right than on the left. This is undoubtedly due to uncomplicated silicosis, probably arising from high concentration of large quantities of dust. The subsequent films made in 1949 with an interval of five years shows extensive progression of emphysema, elevation of both lung roots and the massive lesions are now apparently more dense and larger than they were four years previously. The other subsequent film made in 1959 shows very little change when compared with the 1949 films.

2b



2c



3c

Code - L,J Fig. 3, a, b, c.

The first film made in 1944 shows questionable evidence of uniform nodular fibrosis after 21 years exposure to silica. Subsequent films made in 1951 show an increase in the amount of emphysema and definite evidence of early nodular fibrosis with minimal pleural reaction in the right and left second interspaces. The 1958 and 1959 films still show an increase in the density of both upper lobe areas but no evidence of massive lesions. The nodular fibrosis is slightly more pronounced on the final film than it was on the original film but there is no evidence of complicating infection.

Practical Evaluation of the

Function of the Lungs

• GEORGE R. MENEELY, M.D.
Associate Professor of Medicine and Director
Radioisotope Center, School of Medicine
Vanderbilt University
Nashville, Tennessee

Patients referred for pulmonary function testing usually have, or are suspected of having, chronic pulmonary disease. In industry the situation is different. Here the nature of the employment may be such that induction of disease is a hazard and the purpose of the testing is to identify early changes. The general purpose of testing is to detect impairment and if it is present to characterize it and estimate its severity. The specific purposes derive from the nature of the problem which the particular patient presents to his own physician or to interested third parties such as insurance companies or compensation boards. Procedures suitable for one purpose may not be appropriate for another. In a laboratory primarily organized to provide a clinical service, especially one oriented toward industrial medical problems, instruments must be selected on the basis of versatility. The level of technical skill obtainable is a limiting consideration and the time involved in each test is critical. Personnel costs usually become the major budget item. The efficiency of testing is of the utmost importance. For example, a more expensive instrument which can perform a test more rapidly or more easily would be wise in the long run.

The simplest device which can genuinely be called pulmonary function testing equipment is a spirometric device for measuring the vital capacity and its time relations. From this one can obtain the one- two- and three-second vital capacity, the total vital capacity, the total vital capacity time and, if a tracing is recorded, one can measure it in several other ways described later. Many more tests are possible with the so-called respirometer, a two speed recording spirometer with a ventilograph. The most important features of this instrument are low airway resistance and low inertia of the measuring bell and recording system. The ventilograph invented by Dr. Philip Reichert simplifies calculation of volume ventilation. With the recording respirometer instrument respiratory rate, tidal volume, expiratory reserve volume, inspiratory capacity and minute ventilation at rest and during exercise, as well as maximal voluntary ventilation, the so-called maximum breathing capacity, can be performed. Further, the respiratory patterns can be examined. Oxygen consumption can be measured but during steady states only. This latter datum makes possible such derivative figures as the oxygen removal ratio and the basal metabolic rate. The breathing reserve and the ratio of breathing reserve to maximum breathing capacity can be calculated.

If a helium analyzer is added to the recording respirometer it becomes possible to measure the functional residual capacity and from this and the expiratory reserve volume to compute the residual volume. From the residual volume and the vital capacity the total pulmonary capacity may be obtained. Finally, residual volume may be expressed as a ratio to total lung capacity, usually given in percent. If a blower is used instead of valves, intrapulmonary mixing can be characterized. Certain laboratories make use of open circuit equipment. Our own preference is for the closed circuit methods. If blood gas analysis is to be performed, the traditional Van Slyke manometric apparatus is still the preferred instrument although it is beginning to appear that gas chromatographic methods may well be within the reach of the applied physiology laboratory. Arterial hydrogen ion concentration (pH) can be measured with the glass electrode and if the carbon dioxide content of blood is known from Van Slyke manometric measurements, the arterial carbon dioxide tension can be computed. As will be seen later, this is a valuable figure.

Before directing our attention to what pulmonary function tests can do, it would be well to emphasize what pulmonary function tests cannot do. They cannot provide an etiological or an anatomical diagnosis, giving only estimates of functional capacity and, sometimes, information relative to prognosis. They cannot localize a process geographically, and fail to reveal the location of the lesion even though they may have demonstrated it. They cannot distinguish between processes producing certain effects; for example, it might be possible to demonstrate impaired diffusion but without distinguishing whether it were due to alveolar edema or to interstitial edema. They cannot reveal pulmonary diseases unless function is impaired and then only when impairment is of a sufficient degree. Localized disease may be quite extensive without impairing function whereas diffuse disease is likely to produce functional changes. Pulmonary function tests cannot supplant a careful history, physical examination, and fluoroscopic and radiological examination, nor can they supplant bacteriological or pathological studies. It is important to emphasize that no single test now known is sufficient for evaluation of all aspects of pulmonary function nor is there any single test which can be relied upon absolutely as a screening procedure to identify those patients in whom further studies ought to be done. We will return to this point later. One difficulty with pulmonary function tests is the relative scarcity of information about groups of healthy persons to establish with certainty the normal values of the various pulmonary functions. Many of us are looking to the United States Veterans Administration which is engaged in an immense study on normal subjects which should be of the utmost value. The lack of such data is critically important in industrial applications. It is obvious that any evaluation of industrial hazards and of the effects of noxious agents of industrial origin on the pulmonary function of the individual should distinguish clearly those changes ascribable to the industrial injury from those which are attributable to advancing age or to chronic nonspecific infection unrelated to the industrial exposure.

There are certain things which pulmonary function tests can do which cannot be done otherwise. In the first place they provide objective information for the appraisal of pulmonary impairment, insufficiency and disability when they are interpreted in relation to the other findings in the patient. It is well known that extensive involvement of the lungs may be seen in x-ray shadows which may yet be associated with little or no pulmonary impairment. On the other hand, patients showing little x-ray evidence of pulmonary involvement may have serious pulmonary disability. Pulmonary function tests are useful in ruling out pulmonary disease in nonorganic types of dyspnea such as psychoneurosis, neurocirculatory asthenia or malingering. To a certain extent pulmonary function tests may aid in following objectively the course of pulmonary disease and permit evaluation of therapeutic measures whether medical or surgical. They aid in differentiating cardiac from pulmonary disease and aid in differentiating primary from secondary types of polycythemia. Sometimes they permit detection of abnormalities in subjects whose physical examination or x-ray studies do not disclose pulmonary difficulties. They certainly should be used in pre-employment evaluation. Pulmonary function tests are valuable as an aid in evaluating disability claims in insurance or industrial practice. They are particularly useful because they indicate the specific function of the lung that has been impaired by disease and they give the physician a clearer concept of the processes in each patient.

Normal values for an individual may be predicted from tables or from graphs or from equations, usually based upon height, weight, age, sex, or similar information. The observations made on a patient may be expressed as a percent of the predicted value or as a percent difference from the predicted value.

Respiration is the gaseous exchange between an organism and its environment. It has two major subdivisions. First, internal respiration is the exchange of gases between tissue cells and the fluid bathing these cells. Second, external respiration is the exchange of gases between the blood and the air breathed by the patient. When we discuss pulmonary function we are concerned entirely with external respiration. The mechanisms of external respiration may conveniently be divided into four categories. *Ventilation* is the movement of air in and out of the lungs. *Mixing* is the intrapulmonary mixing of the respiratory gases. *Diffusion* is the exchange of oxygen and carbon dioxide across the alveolar capillary membrane. *Perfusion* (sometimes called circulation) is the quantity and distribution of blood flow through the lungs.

It is of great importance to clarify three words which are commonly misused: impairment, insufficiency, and disability. These have specific technical meanings in clinical physiology. Impairment means a significant deviation of a specific function from normal. Insufficiency means a failure to meet normal requirements for ordinary activity or the necessity for abnormal compensation to meet normal requirements. Disability means the individual is rendered incapable of meeting the requirements of ordinary

activity. Impairment is thus seen to be a laboratory technical matter. One takes the observation and compares it with normal standards. If, on the basis of statistical considerations, it deviates significantly from the normal standards, one speaks of this function as impaired. The concept of insufficiency is quite different. Here the manifestation is no mere statistical matter. Rather, a function is impaired to such a degree that the individual suffers from lack of the benefits of the function. Disability has to do with the relation of the individual to the requirements of ordinary activity. In effect then, impairment has to do simply with deviation of a function from normal, while insufficiency pertains to the effects of this deviation upon the individual. Disability has to do with the degree to which impairment and insufficiency interfere with the individual's meeting of the requirements of ordinary life.

The subdivisions of the lung volume (Figure 1) are the tidal volume, the inspiratory capacity,

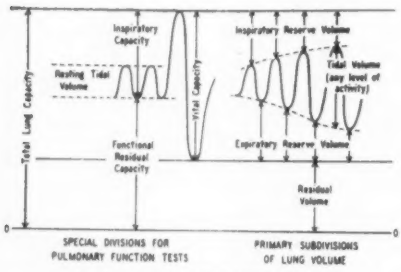


Figure 1

the expiratory reserve volume, the functional residual capacity, the total lung capacity, the vital capacity and the residual volume. As a practical matter the residual volume is derived by subtracting the expiratory reserve volume from the measured functional residual capacity.

Measuring the speed of performance of the vital capacity is more important than measuring the volume of the total vital capacity. It is the simplest method for differentiating obstructive and restrictive ventilatory insufficiency. There are many sorts of equipment available for this purpose. The basic essential in the timed vital capacity measurement is to obtain the shape of the maximal forced expiratory curve. One can measure the one, the two and the three-second vital capacity and express each of these as a fraction of the total vital capacity observed or as a fraction of the total vital capacity predicted to be normal for the individual. Several other measures are applied to this same tracing. Some feel that the most representative portion of it is the middle half. This can be measured either in terms of time, the so-called mid-half vital capacity time, or it can be measured as a flow rate, namely the liters per second expired from the end of the first quarter to the end of the third quarter of the maximal expiratory effort. Sometimes a reciprocal figure is given in which the time to expel a liter of air during the mid-half of the vital capacity is used. Others feel that the peak expiratory flow rate may be the best; that is, the highest velocity obtained at any time during the maximal forced expiration. This usually occurs a few tenths of a second after the initial effort. Still others feel this has drawbacks and prefer to measure the flow rate of the first liter after the first 200 milliliters are expelled. Finally, there are methods which evaluate the last part of the curve, particularly those which involve the comparison of volume and velocity. As a practical proposition, the flow rated during the mid-half of the vital capacity and the three-second vital capacity seem to us to be the most useful screening tests. Some evidence on this subject will be presented later. Comparing various fractions of the vital capacity with the maximum breathing capacity expressed as a percent of the predicted normal for the subject, it reveals that the total vital capacity correlates very poorly. The one-second vital capacity correlates the best; the two-second the next best; and the three-second the next best. From this one would suppose that the one-second vital capacity would be the most worthwhile measure. However, it is difficult technically to be sure of accurate measurements of one-second vital capacity and for this reason most prefer to rely on the three-second vital capacity. It is for the same reason that the velocity with which one liter is expelled after the first two hundred milliliters of air are breathed out has come to be regarded by some as the most useful figure.

The most important feature of the restrictive type of ventilatory insufficiency is that, while the total vital capacity is usually diminished from predicted, the rate at which the test is performed is normal. Ninety percent or more of the observed total vital capacity is expelled in three seconds (Figure 2). In contrast, in obstructive ventilatory insufficiency, while the total vital capacity is also usually less than normal, a fraction usually much smaller than 90% is expelled in three seconds (Figure 3). It is frequently useful to test the total and timed vital capacities before and after bronchodilators for the purpose of evaluating the extent to which reversible bronchospasm plays a role in the subject's problem. Dr. Gaensler summarized some of the clinical alterations of the three-second vital capacity (Figures 4A and 4B). There are

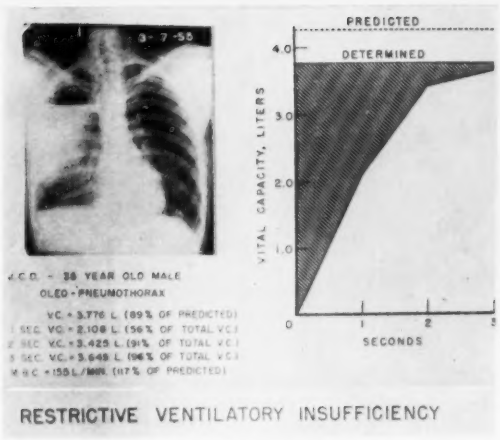


Figure 3A

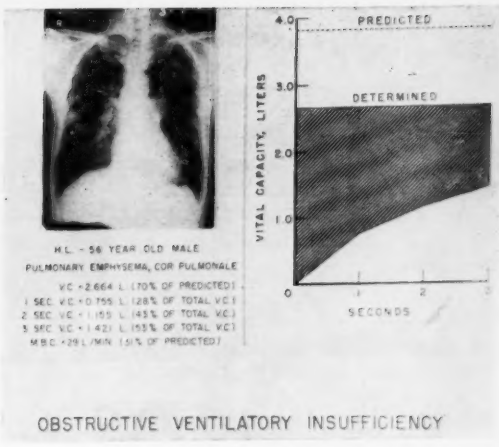


Figure 3B

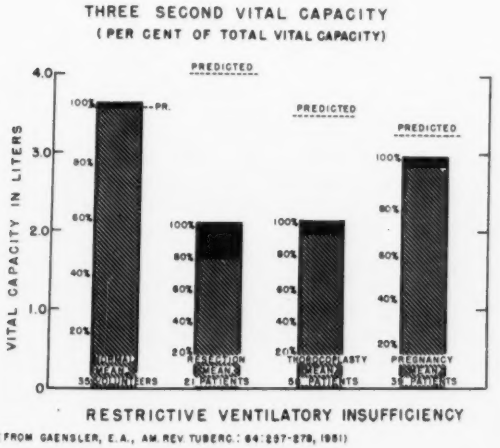


Figure 4A

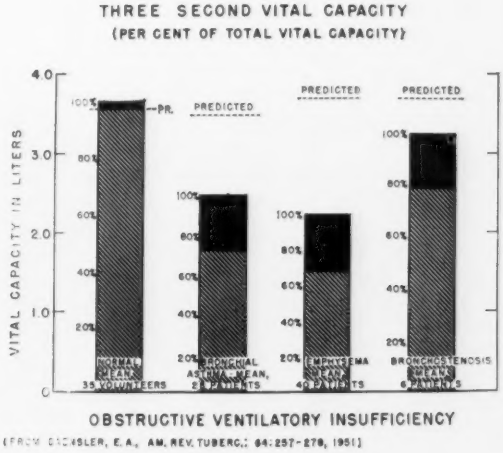


Figure 4B

those who would not agree with his findings in pregnancy. Most find the vital capacity normal or increased in pregnancy despite the large abdominal tumors.

A closed circuit respirometer with a kymograph capable of running at two speeds, namely one-half millimeter per second and two and one-half millimeters per second, permits quite elaborate ventilatory function tests. The ventilograph pen records the total of expiratory volumes in a fixed ratio to the spirographic pen. Spirograms are recorded data that can be compared with other spirograms from the past, or with future spirograms and progress can be evaluated. Spirograms in normals from day to day and week to week are uniform for the individual although they may vary a good deal from person to person. In studying breathing patterns with the carbon dioxide absorber in place, the oxygen consumption can be measured from the slope of the base line. Tracings may help in evaluating the patient's cooperative efforts and his general pattern. They may show hyperventilation or irregular breathing. After running the instrument for a few minutes to record the breathing patterns the individual is instructed to perform a maximum inspiration followed immediately by a maximal expiration, the so called one-stage vital capacity. The two-stage vital capacity is obtained by adding separate values for the inspiratory capacity and for the expiratory reserve volume and it equals the one-stage vital capacity in the normal. In emphysema it is larger than the one-stage vital capacity by a significant amount. Bronchodilators may increase inspiratory capacity and reduce expiratory reserve volume, thereby demonstrating some degree of reversible bronchoconstriction. The vital capacity measurements should be run at least at two and one-half millimeters per second for accuracy of

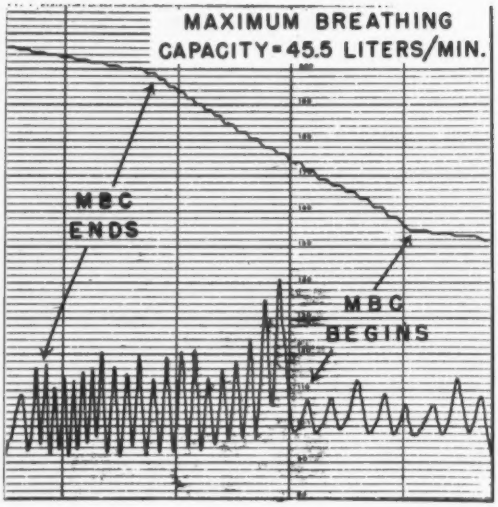


Figure 5A

measurement, and some prefer to use twice this paper speed. Air trapping is best demonstrated by three determinations of the vital capacity performed one immediately following the other. When trapping is prominent each successive vital capacity becomes smaller. If this is due to obstruction which can be relieved, bronchodilators will change the pattern toward normal.

The maximum breathing capacity can best be performed with the closed circuit respirometer. The volume per minute can be measured from the ventilograph tracing and the rate can be measured by counting the cycles on the paper. Normally, the maximum breathing capacity is performed both above and below the base line of the resting breathing pattern. Obstruction and loss of elasticity causes the maximum breathing capacity to be performed at higher levels to overcome the obstruction and to use what elasticity is still available. If obstruction is temporary as in asthma or in certain patients with emphysema, bronchodilators will relieve it, thereby increasing the maximum breathing capacity and characteristically lowering the level at which it is performed. Doctor Guy Hannon has laid particular emphasis on the merits of performing the maximum breathing capacity at a rate timed with a metronome. By this means he is able to obtain more reproducible values from time to time on the same subject and is able more clearly to evaluate tapering of the tidal volume during the MBC which suggests the induction of bronchospasm by the forced breathing itself. In his hands this has been a valuable and reliable test for use in industry. A maximum breathing capacity tracing of a normal subject (Figure 5A) and a patient with

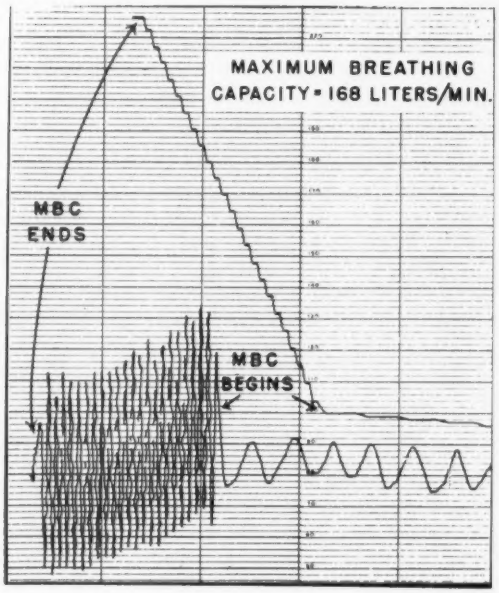


Figure 5B

a markedly diminished maximum breathing capacity (Figure 5B) performed just slightly below the level of the normal breathing pattern, suggests that the patient's defect was of the fibrotic or restrictive variety. An intended normal had the MBC tapering characteristic of bronchospasm induced by forced ventilation (Figure 6). This is an early evidence of pulmonary abnormality.

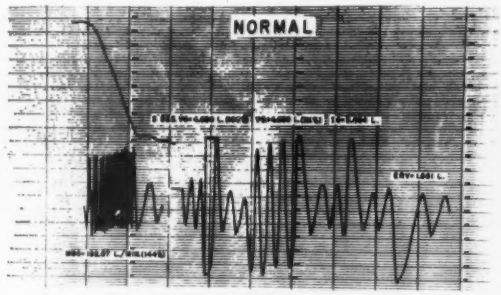
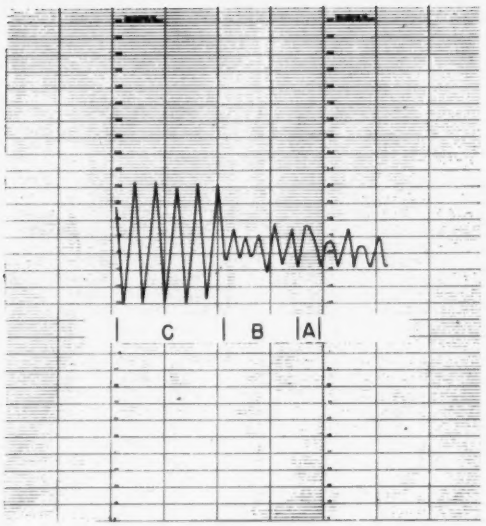


Figure 6

Malingering may be detected in respiratory tracings by the inability of the individual to reproduce the same pattern on several occasions. Despite the fact that motivation plays a part in almost all tests of pulmonary function, it is a curious fact that the values obtained by the tests in common use are remarkably reproducible. A malingerer was requested to perform the one-stage vital capacity test and a voluntary



MALINGERING:

A - Vital Capacity

B - Voluntary Maximum Breathing Capacity

C - Reflex (CO_2) Maximum Breathing

Figure 7

maximum breathing capacity masked. However, when stimulated with carbon dioxide he had a substantial minute ventilation (Figure 7). Un-

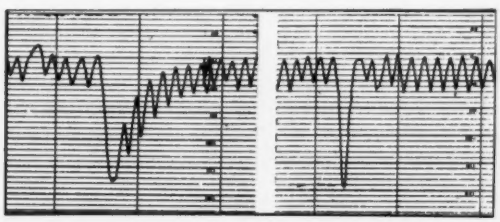


Figure 8A

less a breathing pattern is consistent from time to time undue attention should not be paid to it. An individual seemed to climb down from his maximal inspiratory position in the right-hand

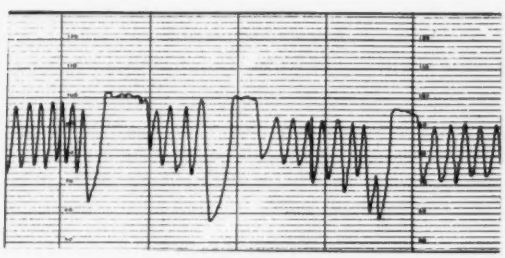


Figure 8B

tracing and one might have wondered what this could mean, but the left-hand tracing shows that the individual is perfectly capable of normal pattern (Figure 8A). Neurocirculatory aesthenia results in erratic tracings. Characteristic of this disorder is a changing base line in quiet breathing and frequent deep sighing. Marked deformity of the chest will, of course, distort the breathing pattern. In an interesting tracing obtained from a patient with Marie-Strumpell disease (Figure 8B) the breathing pattern was basically normal except that the individual could not inspire much beyond the upper level of the resting tidal volume by reason of the rigidity of the spine and the secondary rigidity of the rib cage. It must also be kept in mind that impairment of the heart will produce impairment of the lungs and a consequent distortion of the respiratory tracing and function tests.

Restrictive ventilatory insufficiency which, as earlier remarked, was known in the older nomenclature as the fibrotic defect, is characterized typically by loss of total lung volume with a decreased vital capacity without significant slowing of expiration. Thus, the three-second vital capacity is 90% to 100% of the total observed vital capacity. The maximum breathing capacity may be normal but more often is decreased. When it is reduced, the reduction is disproportionately less than the decrease in vital capacity. The base line at which the maximum breathing capacity is performed is not elevated. This again emphasizes that inspection of the respiratory tracing is a definite superiority of the closed circuit method over open circuit methods for evaluating the simple ventilatory tests. Ventilatory function among patients with restrictive respiratory defects are characteristic (Figures 9A, 9B, and 9C).

Figure 9A

Figure 9B

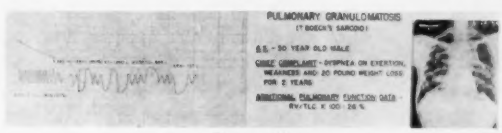


Figure 9C

Figure 10A

Figure 10B

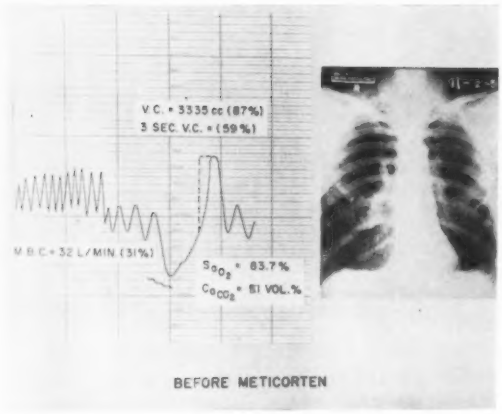


Figure 11A

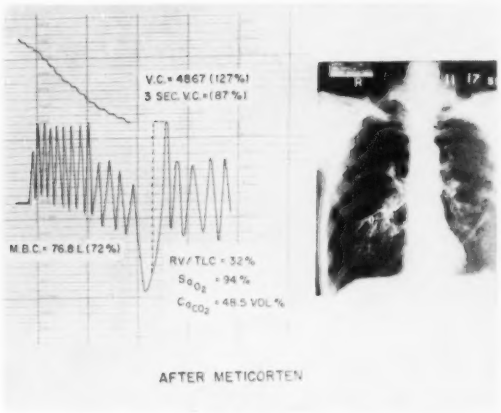


Figure 11B

Obstructive ventilatory insufficiency produces definitive spirographic patterns. Typically, the total vital capacity is decreased although occasionally it may be normal. The important feature is the prolongation of the expiratory time. The three-second vital capacity is less than 90% of the observed total vital capacity and, as earlier remarked, the two-stage vital capacity is greater than the one-stage. The maximum breathing capacity is decreased and performed above the resting base-line level. This elevation of the base-line is a cardinal characteristic feature of obstructive ventilatory insufficiency. It cannot be observed with open circuit methods unless the cumbersome bag and box device is utilized. In the older terminology those who present the obstructive defect would have been characterized as presenting an emphysematous defect. Both terms are unhappy choices and both are inexact. The laboratory diagnosis of pulmonary emphysema has three requirements. First, there must be chronic pulmonary distention as evidenced by an increase in the ratio of residual volume to total lung capacity. While this is obligatory it is not by itself sufficient. There must also be some evidence of slowing of forced expiration and evidence of impaired intrapulmonary gas mixing. Figures 10 and 11 exemplify the obstructive ventilatory defect and the influence on it of certain forms of therapy.

If a helium analytical cell is added to the closed circuit respirometer, it becomes possible to measure the functional residual capacity. From this and the expiratory reserve volume one can determine by subtraction the residual volume of the lung. By adding the vital capacity to the residual volume one obtains the total lung capacity. It is customary to express the residual volume of the lung as a percent of the total lung capacity. Various standards exist for interpretation of this ratio. One which has proved useful in our hands is that the normal ratio of residual volume to total lung capacity is 20 until age 40 and thereafter one-half the age with the range of normal ± 40% (Figure 12). The upper limit of normal for the ratio residual volume to

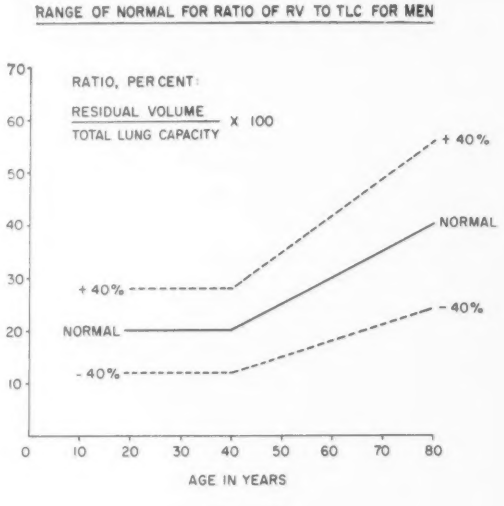


Figure 12

total lung capacity is thus about 28 until age 40 and thereafter rises gradually to 56 at age 80. This rather empirical standard includes most of the published normal data. Some use as the upper limit of normal 35% irrespective of age. This is certainly excessive for young men and probably too strict for older men. On the other hand the standard earlier described may be somewhat too generous for the oldest age group. It is well known that the ratio of residual volume to total lung capacity is greater in women than in men, but the reason is not known.

One question which always arises is what position should the patient assume when the lung volume and its fractions are measured. It is certainly true that individuals standing up can perform better than individuals sitting down and that individuals sitting down can perform better than individuals lying down, i.e., can produce larger volumes. Those of us who are concerned with pulmonary function testing among hospital patients are frequently confronted with a need to make measurements in patients for whom standing is inappropriate. I personally prefer to make all lung volume measurements with the patients semirecumbant with the head of the bed or the examining couch tilted up to an angle of 15° to 25°. This is a position which can be assumed by all subjects capable of performing the tests at all. The most important thing is to perform the tests always in the same way.

Some years ago we introduced the closed circuit helium dilution method for the measurement of the functional residual capacity using the rather complicated technique of replacing the oxygen consumed by the subject. We have now been able to show that the reasons for doing this are unimportant and it is perfectly practical to measure the functional residual capacity with the ordinary closed circuit respirometer with a helium analytical cell added to the circuit (Figure 13). All of the published methods currently in vogue for FRC give results which are sufficiently good to be useful, particularly when one realizes that the ratio of residual volume to total lung capacity is probably the single most important statistic obtained. Thus the nitrogen washout method, the closed circuit helium method, the whole body plethysmographic method and several others are equally useful, although, in our opinion, the closed circuit helium dilution method is the simplest and most advantageous, particularly since the equipment is useful for other purposes as well. Under research laboratory conditions with skilled technicians and careful supervision the standard deviation of the difference between duplicates by the closed circuit helium dilution method is about ± 75 millimeters. Under field laboratory conditions it is perhaps twice this. The over-all effect is that under research conditions and among patients with functional residual capacities less than five liters the error of the mean of a pair of duplicate determinations at the 95% point is approximately ± 54 milliliters and ± 100 milliliters under field laboratory conditions. When the functional residual capacity is very large, in excess of five liters, the error of the mean of the pair of determinations at the 95% point may range up to ± 136 milliliters. It can easily be shown that these variations have little effect on the significance of the ratio of residual volume to total lung capacity. If a closed circuit helium instrument with a blower is used it is possible to evaluate intrapulmonary gas mixing at the same time the functional residual capacity is measured. We have recently developed a technique for characterizing mixing by a single constant based on the entire mixing curve.

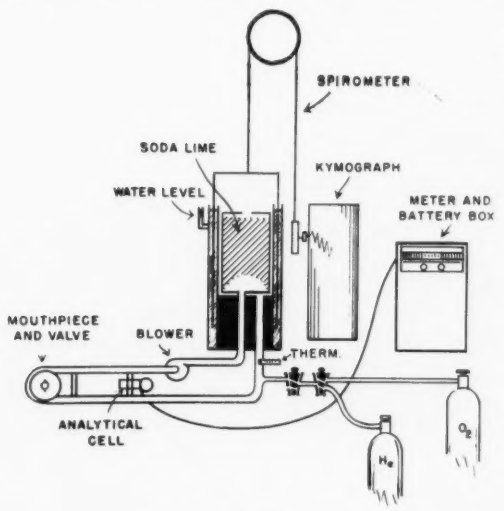


Figure 13

Determination of the arterial oxygen saturation is useful in the elevation of patients with pulmonary disease although it is not usually necessary to measure it in apparently healthy workmen. The primary factors which govern the arterial oxygen saturation and likewise the factors which may be evaluated by this determination are: ventilation, intrapulmonary gas mixing, diffusion and perfusion. Measurement of the arterial oxygen saturation is useful as an over-all test of the four factors listed above, but will not delineate specific impairments of these functions. Combined with other studies it is helpful in the separate evaluation of these four factors. More elaborate blood gas studies are necessary for specific purposes, especially to separate diffusion and perfusion defects. As

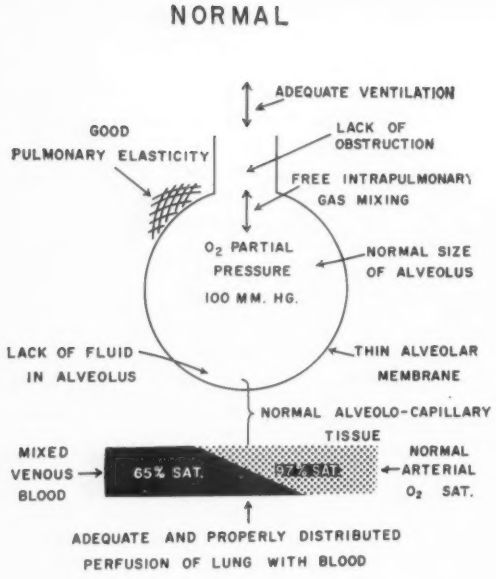


Figure 14

a practical proposition, if an individual's arterial oxygen saturation is normal at rest and remains normal with exercise, it can be stated flatly that he does not have a significant diffusion or perfusion defect. If there is reason to believe that he does have a diffusion or perfusion defect, it can be a major undertaking to distinguish between the two and requires elaborate methods unsuitable for routine work. Although the arterial oxygen saturation is useful in assessing the ability of the lung to oxygenate blood, it is not the most sensitive measure of this function. More elaborate blood gas tension studies may reveal impaired diffusion in some patients who have normal arterial oxygen saturation. Let me warn that, at the present time, ear oximeter methods are still to be regarded as in the developmental stages. There are some serious drawbacks to the use of the oximeter. Until tests involving the ear oximeter have been more widely explored it would be well to regard them as primarily research procedures.

Normal saturation of the arterial blood with oxygen (Figure 14) is dependent upon adequate ventilation, good pulmonary elasticity, absence of airway obstruction, free intrapulmonary gas mixing, normal sized alveoli, absence of fluid from the alveoli, a thin alveolar membrane, normal alveolar capillary tissue, and adequate and properly distributed perfusion of the lung with blood. Impaired ventilation characteristically causes a reduction in the partial pressure of oxygen in the alveolus. It may be due to depressed respiratory center, to impaired respiratory mechanics, to impairment of the diaphragm, to any other restriction of mobility of the lungs, to obstruction of the respiratory passageways, to impaired pulmonary elasticity, and to chronic pulmonary distention. Impaired intrapulmonary gas mixing is usually due to obstruction, most commonly in the smaller bronchial ramifications, and also may result in a lowered partial pressure of oxygen in the alveolus. All of the factors previously mentioned as concerned with impaired ventilation also apply to impaired intrapulmonary gas mixing. In addition, important are

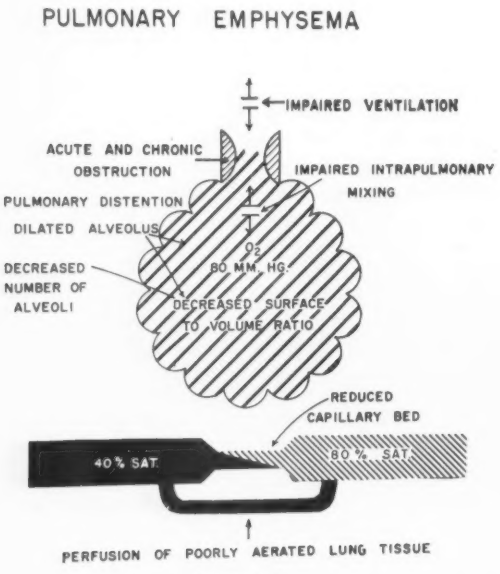


Figure 15A

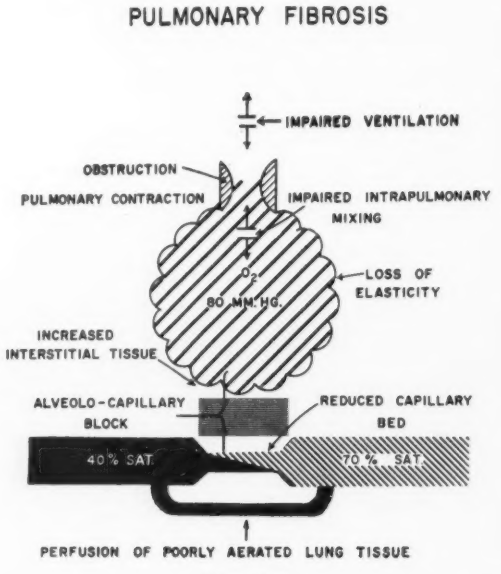


Figure 15B

emphysematous bleb formation, air cysts and bronchiolar obstruction. In chronic pulmonary disease these factors are usually progressive. For diffusion of gas from the alveolus to the capillary blood there are five layers which must be traversed. First, the alveolar membrane; second, the interstitial fluid; third, the capillary membrane; fourth, the plasma; and, fifth, the red cell. Disease affecting any of these elements may result in apparent impairment of diffusion. Fluid in the alveoli and/or a thickened alveolar wall and/or interstitial thickening all produce a picture commonly called alveolocapillary block. It is characteristically seen in fibrosis or inflammation of the pulmonary parenchyma. Important perfusion (impaired circulation) has the effect of a pulmonary arteriovenous shunt. When blood is distributed to nonaerated or poorly aerated lung, some of the poorly oxygenated pulmonary arterial blood gains direct access to the pulmonary veins and thereby dilutes the well oxygenated pulmonary venous blood.

In the usual case of pulmonary emphysema with arterial oxygen unsaturation, no single function is fully responsible for the impairment of oxygenation. Usually ventilation, intrapulmonary gas mixing and perfusion are all impaired. Determination of the arterial oxygen saturation in pulmonary emphysema is frequently used in combination with the measurement of arterial carbon dioxide content for classification of the severity of the disease. This is especially true when measurements are made after exercise as well as at rest. In addition, serial determinations of arterial oxygen saturation in any given patient with pulmonary emphysema are helpful in determining the type of therapy required and in evaluating its effectiveness. In pulmonary emphysema the physiological defects can be summarized as: impaired ventilation, acute and chronic obstruction, pulmonary distention, dilated individual alveoli, a decreased number of alveoli and a decreased surface to volume ratio of the remaining alveoli (Figure 15A). Further, there is impairment of intrapulmonary gas mixing and a loss of pulmonary elasticity. There is a reduced capillary bed and perfusion of poorly aerated lung tissue with blood thereby resulting in shunting. Bronchiolar narrowing with accumulated secretion, mucosal thickening, edema, and varying degrees of bronchospasm and bronchoconstriction occur. Other elements of impaired mechanics of breathing in emphysema must be recognized, notably flat diaphragm with resulting loss of piston effect and inspiratory lower rib retraction. Further, the hyperinflated immobile chest cage is disadvantageous mechanically and the accessory muscles often fail to relax. There is a general incoordination of the muscular effort and of breathing and, commonly, disadvantageous breathing habits are acquired by the patient.

In pulmonary fibrosis there may be much variation in impairment of pulmonary function (Figure 15B). The arterial oxygen saturation in patients with different types of pulmonary fibrosis is highly variable. In those cases of pulmonary fibrosis which lead to airway obstruction, pulmonary emphysema also is frequently found. Then arterial oxygen saturation will be influenced not only by the basic lesion but also by the factors discussed under pulmonary emphysema. In some patients with pulmonary fibrosis, diffusion of gases across the alveolocapillary membrane is the only significant lesion, the so-called alveolocapillary block syndrome. Measurement of the arterial oxygen saturation may be most helpful here for it may be the only commonly available measurement which reveals significant impairment. There are patients with evident pulmonary fibrosis with no impairment of oxygenation of the blood so a normal arterial oxygen saturation value may also be helpful. In summary, the leading features of pulmonary fibrosis from the pulmonary physiological point of view are: impaired ventilation, airway obstruction (both reversible and irreversible), pulmonary contraction, impaired intrapulmonary mixing of gases, reduced alveolar oxygen partial pressure, loss of pulmonary elasticity, increased interstitial tissue with resulting alveolocapillary block, a reduced capillary bed and perfusion of poorly aerated lung tissue resulting in venous admixture.

Proper carbon dioxide elimination is as important a function of respiration as oxygenation. Arterial carbon dioxide tension (the partial pressure of carbon dioxide) is a measure of free carbon dioxide while pH is the measure of relative acidity or alkalinity of the blood. Since carbon dioxide is highly diffusible, the carbon dioxide tension of blood leaving the pulmonary capillary is the same as the carbon dioxide tension in the alveolus. Alveolar carbon dioxide tension therefore controls arterial carbon dioxide tension even in quite advanced pulmonary impairment. Alveolar carbon dioxide tension in turn depends upon ventilation and intrapulmonary gas mixing.

There are many methods to measure arterial carbon dioxide tension and arterial pH. The easiest adequate method depends upon certain relations which exist between arterial pH, carbon dioxide content and carbon dioxide tension. The carbon dioxide content of a blood sample can be measured with the Van Slyke manometric apparatus and pH measured directly with the glass electrode pH meter. From this information

arterial carbon dioxide tension can be calculated.

Factors affecting the control of ventilation which are of greatest concern in pulmonary function testing are arterial carbon dioxide tension, pH, oxygen tension and muscular reflexes. Increases in blood carbon dioxide tension normally result promptly in stimulation of ventilation and elimination of the excess carbon dioxide. Inadequate ventilation for whatever reason results in carbon dioxide retention. If carbon dioxide retention persists, renal retention of base restores pH to normal, thus compensating for the high carbon dioxide tension. Since retention of base is a feature of chronic carbon dioxide retention and because pH is usually maintained close to normal, the plasma carbon dioxide combining power (which is a measure of plasma base) is increased in close proportion to the degree of chronic carbon dioxide retention. Thus the plasma carbon dioxide combining power is one of the guides to therapy of patients with inadequate ventilation. This is particularly useful because plasma carbon dioxide combining power determinations are usually obtainable on an emergency basis in most hospitals whereas other sorts of blood gas analysis may be more difficult to obtain. The usefulness of this measure depends upon the fact that the hydrogen ion concentration of the blood (pH) rarely varies outside of narrow limits and can be assumed to be close to normal in an individual unless he is unconscious or near death. If the carbon dioxide combining power (the fixed base) of plasma is high and the pH is near normal as it must be, then the carbon dioxide tension must be high. In other words, such a patient probably has carbon dioxide retention and is therefore a candidate for carbon dioxide narcosis if anything is done to cause a decrease in ventilation.

With normal ventilations and intrapulmonary gas mixing, the partial pressure of carbon dioxide in the alveoli is usually 40 millimeters of mercury. Normally, there is no significant alveolar-arterial blood carbon dioxide gradient. Thus, in the blood in any given pulmonary capillary, the partial pressure of carbon dioxide will be the same as the partial pressure of carbon dioxide in the adjacent alveolus. With normal alveoli and pulmonary capillaries throughout the lung, the partial pressure of carbon dioxide in arterial blood and therefore also in 40 millimeters of mercury will be the same. With a normal amount of blood buffer base, the pH will then be normal.

Emotions, drugs, central nervous system disorders, high altitudes and other factors may cause hyperventilation. With hyperventilation excessive carbon dioxide is blown off and the alveolar carbon dioxide tension is lowered. Acutely, this results in a decreased arterial carbon dioxide tension and increased hydrogen ion concentration. This situation is known as respiratory alkalosis. With prolonged hyperventilation, renal loss of base lowers the pH and leads to a compensated respiratory alkalosis. Prolonged hyperventilation rarely occurs as a result of chronic pulmonary disease but is of importance in understanding pulmonary function in certain situations; for example, in patients placed under artificial respiration in respirators.

Inadequate ventilation for any reason results in an increase in the partial pressure of carbon dioxide in the alveolus. Since the arterial blood partial pressure of carbon dioxide is in equilibrium with the alveolar carbon dioxide there will be an increase in arterial partial pressure of carbon dioxide. Acutely, this carbon dioxide retention causes lowering of the pH and is known as respiratory acidosis. If carbon dioxide retention persists, renal retention of base raises pH to normal thereby compensating for the high carbon dioxide tension. This condition is compensated respiratory acidosis.

In chronic pulmonary disease the usual abnormality of arterial carbon dioxide and pH is that which accompanies hypoventilation (Figure 16). There is commonly a respiratory acidosis which may or may not be compensated. In addition, by the time carbon dioxide retention and acidosis occur, anoxia is always also present. In such patients, anoxia is the main stimulus to ventilation because the respiratory center has become insensitive to carbon dioxide. Excessive oxygen therapy may abolish anoxia and further depress

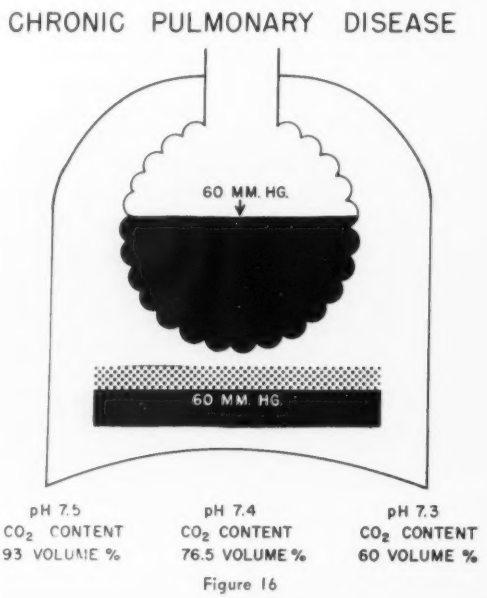


Figure 16

respiration. Further retention of carbon dioxide may occur rapidly and lead to carbon dioxide narcosis and even death if steps are not taken to insure adequate ventilation. Despite this, oxygen therapy is often urgently necessary and should not be withheld. It should, however, be given in such a manner and with such precautions that carbon dioxide narcosis will not occur.

The fluoroscope must be regarded as an instrument for pulmonary function testing. One should fluoroscope each patient in whom pulmonary function studies are done and one should attempt to estimate from the fluoroscopy what one would expect to find with the pulmonary function tests. If there are discrepancies between the findings suggested by a carefully done fluoroscopy and the observations made in the laboratory, particular attention ought to be directed to resolve this discrepancy. This is especially true in bronchospirometric comparison of pulmonary function of each lung. It is inappropriate to our purpose today to discuss bronchospirometry at any length beyond mentioning that a pulmonary physiology laboratory intended to support a surgical chest program should engage in bronchospirometry.

As a late stage in any pulmonary disease, heart disease secondary to pulmonary diseases may well develop. The principal mechanism whereby pulmonary disease produces heart disease is by obstruction to pulmonary blood flow and resulting pulmonary hypertension with the development of right heart hypertensive disease. The contribution of anoxia and disturbed electrolyte and metabolic patterns should not be overlooked as significantly altering matters in an unfavor-

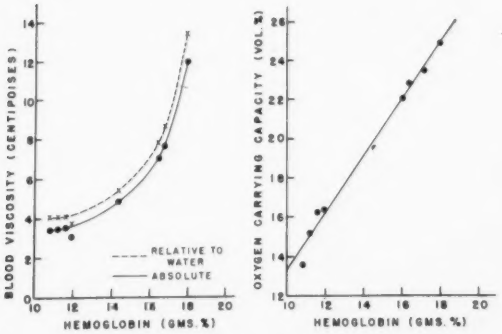


Figure 17

able direction. There is little evidence from pathology that anoxia affecting the left ventricle is a contributing factor in pulmonary heart disease but it certainly is a clinical impression that this must be the case in some patients.

One of the effects of anoxia is to stimulate new hemoglobin formation as manifested by an increase in the volume of packed red cells, the so-called secondary polycythemia of severe pulmonary disease. It should be noted that while the oxygen carrying capacity of blood increases as a simple linear function of hemoglobin concentration, the viscosity of blood increases exponentially (Figure 17). If one studies the effect of this upon hemodynamics, it can be seen that excessive work is required to circulate the viscous blood and thus the gain in oxygen carrying capacity is more than offset by the amount of work which has to be done to force the thickened blood through the small blood vessels. In patients with severe pulmonary disease and severe anoxia accompanied by polycythemia, phlebotomy may be life-saving and often produces a dramatic improvement in what had seemed at first a hopeless or near hopeless situation. It is probable that we do not employ phlebotomy nearly as often as we should. There was gross abuse of phlebotomy as a medical therapeutic measure, particularly toward the end of the last century. This life-saving procedure has frequently not been used when it should because of the general disrepute into which it has fallen.

Some years ago Baldwin, Cournand, and Richards offered a physiological classification of pulmonary emphysema which we have not seen any reason to abandon. Class I is ventilatory insufficiency; no anoxia is present. Class II is alveolorespiratory insufficiency with anoxia but with a partial pressure of carbon dioxide under 48; i.e., no serious carbon dioxide retention has yet occurred. Class III is alveolorespiratory insufficiency with anoxia and a carbon dioxide partial pressure over 48 millimeters of mercury. Class IV exhibits the manifestations of Groups II and III and, in addition, exhibits right congestive heart failure.

At the present time it is not practical to measure pulmonary compliance in most laboratories engaged in routine pulmonary function testing. One should view with some suspicion the simplified methods which are sometimes advocated for this purpose. Despite the fact that this is a procedure which should be reserved to fairly advanced laboratories, it is appropriate to say a word or two about it. Pulmonary compliance is a measure of the elastic property of the lung in terms of the volume change in liters corresponding to a pressure change of one centimeter of water. Specifically, compliance equals change in lung volume in liters divided by change in transpulmonary pressure in centimeters of water. To measure it, one needs to know the volume of the air inhaled or exhaled and at the same time the pressure applied to the lungs, for example, by means of an oesophageal balloon

PULMONARY COMPLIANCE

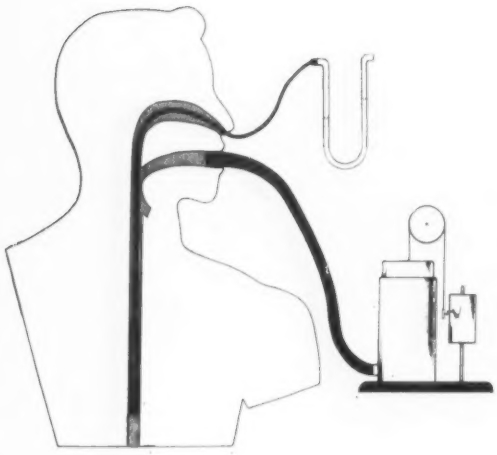


Figure 18A

PULMONARY RESISTANCE

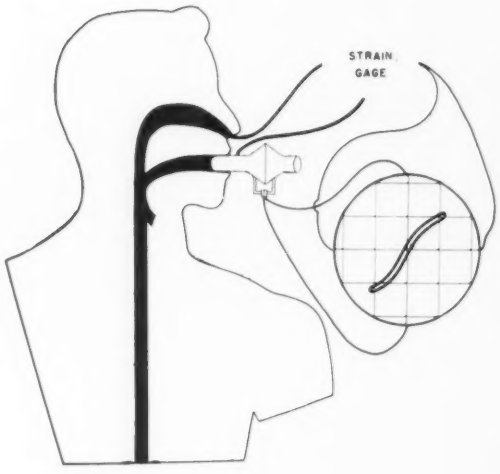


Figure 18B

(Figure 18A). The measure must be made at a time when air flow is zero by this method, that is, at the end of inspiration and expiration. Other methods make possible measurement at other stages of respiration. Compliance is related to the volume to which the lungs are inflated, the architectural pattern of the lungs, the elastic properties of the lungs, the mass of lung tissue, the rate of respiration and the degree of uneven ventilation. Pulmonary compliance is decreased in pulmonary fibrosis, in left heart failure with pulmonary congestion and in cor pulmonale. Static compliance (single breath) is increased in uncomplicated pulmonary emphysema, but when measured at the resting respiratory rate it is decreased (functional compliance). It therefore will vary with the respiratory rate, probably depending on the rate of air flow (pulmonary viscous resistance) and uneven distribution of ventilated air.

Pulmonary resistance is also a measure which can only be made in relatively advanced laboratories. Pulmonary resistance results from the frictional resistance of the airways to air flow and the tissue viscous (plastic) resistance due to the movement of the tissues. Specifically, pulmonary resistance is defined as the pressure in centimeters of water divided by the flow produced in liters per second. It may be measured by obtaining the transpulmonary pressure, namely the pressure between the lungs as measured for example by the oesophageal balloon and the mouth by a strain gauge and simultaneously measuring the rate of air flow by a pneumotachygraph (Figure 18B). Normally, pulmonary resistance is 1.10 to 2.55 centimeters of water pressure per liter per second at air flows of one liter per second. It is most useful for measurement of the degree of obstruction in asthma and emphysema, particularly prior to and following therapeutic effort with bronchodilators, thereby giving a good measure of the efficacy of the therapeutic measure and at the same time an evaluation of the degree to which reversible bronchiolar constriction is important in the particular patient.

Finally, it is appropriate to say a word about right heart catheterization in the evaluation of patients with pulmonary disease. Again, this is a measure available only in fairly advanced laboratories and does not have a role in field testing in industrial medicine. It does provide information which cannot be obtained in any other way to characterize the difficulties of a patient with

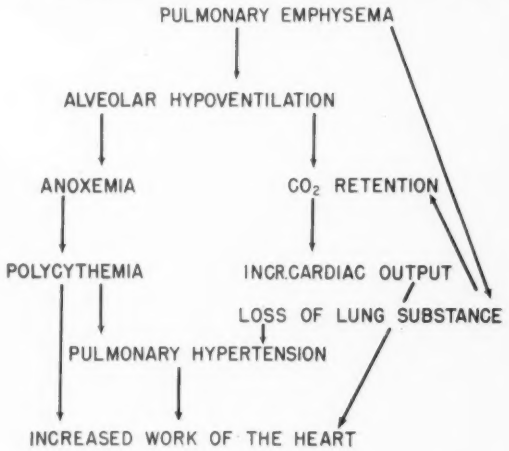


Figure 19

moderate to severe pulmonary impairment who is suspected of having a cardiac factor secondary to his pulmonary disease. The mechanism whereby heart disease is produced in chronic pulmonary disease is fairly straightforward (Figure 19). Pulmonary emphysema, for example, produces two main immediate effects: alveolar hypoventilation and loss of lung substance. Both the loss of lung substance and the alveolar hypoventilation contribute to carbon dioxide retention which in turn stimulates cardiac output thereby increasing the work of the heart. Further, alveolar hypoventilation results in anoxemia. Anoxemia in itself does not help the heart to meet its demand, but makes matters worse by leading to secondary polycythemia. Both polycythemia and the diminished amount of capillary bed by reason of loss of lung substance contribute to obstruction of blood flow through the lung and result in pulmonary hypertension. All this occurs at a time when the heart is stimulated to an excess output. It is possible to characterize the nature of the defect in cor pulmonale rather completely by cardiac catheterization, particularly by means of measuring the pulmonary artery mean pressure, the cardiac output, the right atrial pressure, and the total pulmonary resistance.

For purposes of industrial medicine it may frequently suffice to utilize screening tests and refer those subjects in whom the screening tests are positive for more elaborate studies by a consulting laboratory. We have recently investigated the relative efficiency of various screening methods. Seventy-seven percent of a group of hospital patients had some objective evidence of abnormal pulmonary function. The following screening tests and criteria were tried in this group:

VC (total as % of predicted)	Less than 90%
3 Sec VC (as % of observed)	Less than 90%
3 Sec VC (as % of predicted total VC)	Less than 90%
MBC (as % of predicted)	Less than 90%
Mid-half VC flow rate	Less than 2.9 liters per second
RV/TLC %	Above normal range. (See Figure 12)

The total vital capacity is a quite inefficient screening method, finding only 35% of those detected by other tests. It is negative in 65% of cases when one or more of the other tests are positive. The three-second vital capacity (% of observed), three-second vital capacity (% of predicted total VC), and the mid-half vital capacity flow rate are progressively more efficient, giving positive results in 46, 57, and 72% respectively. The MBC% was also positive in 72% of the patients with one or more other tests positive. As may be seen, however, it still fails to detect pulmonary function abnormality in the 28% remaining. If one combines MBC% and mid-half vital capacity flow rate, only 9% of patients with demonstrated abnormalities escape. If one combines MBC%, mid-half vital capacity flow rate and three-second vital capacity (% of predicted total VC) only 2% escape. In other words, this combination is 98% efficient at finding pulmonary function abnormalities among these subjects. It must be recognized that this efficiency is relative and does not take account of the fact that one or more of these tests might be falsely positive.

In conclusion, it appears from the above that, for screening purposes, the following policy should be effective: three-second vital capacity, mid-half expiratory flow rate and the maximum breathing capacity should be measured in all subjects. Among those found outside the limits specified above by any of these, the lung volume should be measured and the ratio of residual volume to total lung capacity calculated. Timed vital capacity and the maximum breathing capacity can be done in two to five minutes per subject while FRC with RV/TLC done in duplicate requires 30 to 40 minutes and is thus manifestly unsuitable for routine survey purposes. On the other hand, elevated RV/TLC is obligatory for the laboratory diagnosis of pulmonary emphysema although not alone sufficient. It appears from the data above that RV/TLC may be done only in those screened by the three tests recommended. Other studies of our data suggest there may be 6% to 8% of patients with elevated RV/TLC who do not manifest abnormal vital capacity time relations or maximum breathing capacity deficiencies. The significance of this finding is not yet known.

It might be well to conclude this discussion with a look toward the future. Are there methods emerging from the research laboratory which will greatly change the present picture? The answer to this question is probably yes. Preliminary work in many laboratories indicates that the whole body plethysmograph is a powerful tool. At the present time one cannot procure from any single vendor a complete body plethysmographic apparatus suitable for routine clinical pulmonary function testing but it appears that it will not be long before such is available. At the present time the essential components of an adequate plethysmographic laboratory are available and those who are versed in pulmonary function testing should have no difficulty in setting up these methods. Many procedures which can be done only with difficulty or which are time consuming by older methods can be

done easily, rapidly and accurately by the whole body plethysmographic method. Other things which cannot be done at all by currently available routine laboratory equipment are quite practical. All of us in this field are looking with great interest at developments in this line. Ear oximeter methods are advocated by some and are tantalizingly promising. So far, one's attitude should be guarded because of the well known vagaries of the ear oximeter. Some of the methods under development which show promise are those which depend upon the rate at which arterial oxygen changes take place as a subject is switched from breathing room air to a low oxygen mixture then to high oxygen mixtures and then back to room air. When ventilation is taken account of and such measurements are carefully made, dependence upon the rate of change eliminates some of the objections to the inaccuracies of absolute oximetry. Adequate clinical experience with such methods, however, has not yet been accumulated. Finally, it should be reported that promising results are emanating from those laboratories which are exploring volume-velocity loops. These can be obtained with relatively simple equipment. Again, unfortunately, this is not readily available.

Acknowledgements

Much of this material is drawn from the anual American Medical Association Pulmonary Function Testing Exhibit. Thanks are due to Dr. Julius Comroe, Jr., particularly as to what pulmonary function tests can do and cannot do; to Dr. Edwin Gaensler for material concerning timed vital capacity and to Dr. John Seabury for material concerning fluoroscopy. Thanks also are due to those who now and in the past have been associated with me in the actual development of the material for this exhibit, notably Dr. Donald Tysinger, Dr. Oscar Balchum, Dr. Joseph Merrill, Dr. James Callaway, Dr. Lloyd Ramsey and Mrs. Con O. T. Ball.

Research reported was supported in part by the Public Health Service (NIH H-4800 [SSS] and SAph 70219) and the United Fund of Greater Chattanooga.

You Are Getting Sleepy...

Hypnosis is a form of communication and not the result of mysterious forces, says a University of California psychologist, Dr. Theodore R. Sarbin. Speaking before the Department of Psychiatry of the University of Wisconsin Medical School on "Current Concepts in Hypnosis," he described modern science's view and study of this curious state which has puzzled men for several hundred years.

His studies indicated that people under hypnosis could not be made to commit antisocial acts against their will, nor does hypnosis seem to improve learning. But Dr. Sarbin said that almost any kind of physiological process can be affected through communication processes in appropriately predisposed subjects.

Experiments showed that many reactions thought due only to hypnosis could be brought about by conscious suggestion, or by asking people to act as if they were hypnotized.

A hypnotized subject responds to commands from his hypnotizer in exactly the same way that a stage actor responds to commands from his director.

Ability to be hypnotized depends upon prior conception of how a hypnotized person should act. Most of us know what Hamlet's conflicts were, and so we have an idea about how to play Hamlet on the stage. We must likewise have an idea of how a hypnotized person should act before we are capable of becoming hypnotized.

Ability to become hypnotized also depends on motivation — what the subject believes he will get out of it — and on the ability to shift perspective. The ability to shift one's perspective, and thus the ability to be easily hypnotized, seems closely related to a good imagination. This aptitude is frequently found in people who need a reciprocal relationship — lonely people. Those who seek novelty and challenge also tend to be easily hypnotized.

The psychologist said that past experiments seemed to indicate that people could be made to steal under hypnosis, but this idea was the result of experiments done in highly artificial surroundings — in laboratories — where subjects believed that the scientist would not have them do anything wrong.

More recent experiments conducted under normal conditions showed that hypnotized subjects would not steal in surroundings where the moral decision would fall upon themselves.

Experiments showed that the involuntary nervous system could be affected by hypnosis. Hungry hypnotized subjects who were told they were eating showed physiological changes in the stomach which accompany actual eating. This is not a mysterious force. The bridge between hypnosis and physiological changes is emotional excitement and conscious physical acts. The subjects who showed most marked stomach reactions also did the greatest amount of chewing, lip-smacking, and swallowing.

The 1960 Industrial Health Conference

Rochester, New York

April 25-29, 1960

Industrial physicians, dentists, nurses and hygienists arrived in Rochester, New York, to attend sessions of the 1960 national Industrial Health Conference April 25-29. The total number of members of each association attending were as follows: Industrial Medical Association, 673; American Industrial Hygiene Association, 501; American Conference of Governmental Industrial Hygienists, 115; American Association of Industrial Nurses, 474; and American Association of Industrial Dentists, 13. There were 586 scientific and technical exhibitors, wives, and other guests; a total registration of 2,362. General conference sessions were held Tuesday through Friday in the Rochester War Memorial.

At the week-long meeting, industrial health experts and medical directors of leading U.S. companies and governmental organizations heard reports on a broad spectrum of medical subjects.

Scientific papers and panel discussions were presented on heart disease in industry, cancer detection, mental health, radiation safeguards, industrial dentistry, industrial nursing, air pollution, and toxicology of various substances important to the public and to industry.

General conference chairman was HERBERT T. WALWORTH, Director of Industrial Hygiene for the Lumberman's Mutual Casualty Company of Chicago.

Considerable emphasis at the conference was placed on ways to improve preventive medical services provided by industry and on progress in control of occupational health hazards.

Mayor PETER BARRY of Rochester gave a welcoming address to a general session of the conference in the War Memorial. The Sappington Memorial Lecture of the Industrial Medical Association was given by WILLIAM P. SHEPARD, M.D., in the War Memorial. DR. SHEPARD is Medical Director of the Metropolitan Life Insurance Company. His topic was "Industrial Medicine — What It Takes," an analysis of small plants and the general practitioner in industry.

Legal aspects of medical science received attention in several sessions of the conference. These included a demonstration of the role of the industrial nurse at a typical workmen's compensation hearing; a mock trial of a hypothetical case of heart disease in workmen's compensation with leading physicians and attorneys for plaintiff and defendant; and a session considering radiation injury and worker compensation. NOEL S. SYMONS of Buffalo spoke to industrial hygienists on "What Is Expected of the Professional Person in Litigation."

ROBERT B. NEMEROFF, D.D.S., President-Elect of the American Association of Industrial Dentists, presided at a meeting on communication and the exchange of scientific information. The important role of the nation's press in presenting news of medical and dental progress and ways to improve communication among professional groups were among the subjects explored.

Scientific and technical exhibits in the War Memorial supplemented the reports and discussions at the meetings.

A program of tours for the conference delegates and their wives took the visitors to the radiation therapy and other units of the Atomic Energy Commission's installation at the University of Rochester; to the glass plant of Bausch & Lomb Optical Co.; production areas of Delco and Rochester Products divisions of General Motors; to Eastman Kodak Company plants, to Gleason Works, and to Stromberg Carlson Division of General Dynamics. Each tour will illustrate a specific industrial process plus the medical facilities of the particular company.

Wm. P. Shepard, M.D. (right), receives the Knudsen Award for his outstanding achievements in occupational health. The Award was presented by Kieffer Davis, M.D. (left).

Mr. Seaton (right), Vice-President of Personnel, accepts the Health Achievement in Industry Award for General Motors Corporation. The Award was presented by Richard A. Sutter, M.D. (left).

Elections

The Industrial Medical Association elected the following officers for the 1960-1961 term: President — ROBERT E. ECKARDT, M.D., Ph.D., Medical Director of Esso Research and Engineering Company, Linden, New Jersey; President-Elect — GRADIE ROWNTREE, M.D., Medical Director of Fawcett-Dearing Printing Company, Louisville, Kentucky; First Vice-President — H. GLENN GARDINER, M.D., Medical Director of Inland Steel Company, East Chicago, Indiana; Second Vice-President — JOHN L. NORRIS, M.D., Medical Director of Eastman Kodak Company, Rochester, New York; Treasurer — JERMYN F. MCCAHAN, M.D., Medical Director of Western Electric Company, Chicago, Illinois; and Secretary — LEONARD ARLING, M.D., Minneapolis, Minnesota.

Elected to the Board of Directors for the 1960-1963 term were: MERLE BUNDY, M.D., Assistant Medical Director of United States Steel Corporation, Pittsburgh, Pennsylvania; ANTHONY D'ALONZO, M.D., Assistant Medical Director, E. I. duPont de Nemours and Company, Wilmington, Delaware; JEAN SPENCER FELTON, M.D., Professor of Occupational Health, Department of Preventive Medicine and Public Health, University of California School of Medicine, Los Angeles; SEWARD E. MILLER, M.D., Director, Institute of Industrial Health, and Chairman, Department of Industrial Health, School of Public Health, University of Michigan, Ann Arbor; and PAUL J. WHITAKER, M.D., Medical Director of Allis-Chalmers Manufacturing Company, Milwaukee, Wisconsin.

Banquet

The Forty-Fifth Annual Banquet of the Industrial Medical Association was held April 29 in the grand ballroom of the Manger Hotel. D. JOHN LAUER, M.D., retiring President of the Industrial Medical Association, presided over the activities. ARTHUR S. FLEMMING, Secretary of the U. S. Department of Health, Education and Welfare, was unable to deliver his scheduled address owing to other commitments. His place was taken by DR. DAVID E. PRICE of the San Diego Public Health Service, and former Deputy Director of the National Institutes of Health. The Health Achievement in Industry award was presented by RICHARD A. SUTTER, M.D., to the General Motors Corporation.

The William S. Knudsen Award, "industrial medicine's highest honor," was presented by KIEFFER DAVIS, M.D., to WILLIAM P. SHEPARD, M.D., Medical Director of Metropolitan Life Insurance Company, New York City.

Books

First Aid Guide for the Small Plant

Detroit Industrial First Aid Advisory Committee, Alfred H. Whittaker, M.D., Chairman (First Aid Guide, Box 241, Winnetka, Illinois) 80 pp., illustrated, paper, 75 cents (quantity prices on request)

Almost invariably the first person to reach and take emergency care of an injured worker is a layman. What that layman does may determine the magnitude of the services later required of the physician or surgeon. More, it may influence the earthly future of the worker, as well as the extent of the employer liabilities incurred on his account. This makes it imperative that what is done be done promptly and properly; "improper first aid may be ruinous; distinct harm may be introduced by those of good intent but without proper qualifications." Thus the layman must know what to do, and how to do it. This Guide tells him both. It is a layman's First Aid Manual, useful in any work place, though designed primarily for the smaller situations that do not have in-plant medical services or arrangements with outside medical clinics. The instructions are those likely if a physician were giving them. The language is simple and without ambiguities. Nothing applicable to the industrial setting has been omitted; nothing that is not applicable has been included. And the contents are as fresh and new and up-to-date as the latest method in mouth-to-mouth breathing for artificial respiration.

This First Aid Guide was prepared by the Detroit Industrial First Aid Advisory Committee — 14 industrial physicians and surgeons and industrial hygienists who are experienced in the requirements of industrial first aid, and whose names in themselves are assurance of the high order of content. Probably "developed" is a better term than "prepared" to describe the work of the Committee; the Guide has been years in the making. It had its inception in the mid-1940's when the first studies were made toward "a medical emergency plan for small work places." From these studies were evolved the provisions around which the present Manual has grown: (1) a definite plan of procedure in case of serious accident or acute illness among employees; (2) an adequate first aid kit, properly located; and (3) personnel properly trained to use the kit. These provisions were considered minimal for any work place. They were first published in 1950. In 1952 the Committee, having experienced the growing and articulate need for a Manual to be used in training the personnel in (3) above, designed "a course and guide for small establishments, and for which a suitable teaching schedule could be arranged," and that "course and guide" was published the same year. An accompanying editorial comment said: "This may not be the only edition of the Manual." The comment was prophetic; successive editions brought it to its fourth printing in 1956.

During the next three years, the wide use of the Guide and the increasing demand for it among industries, clinics, schools, insurance companies, and adult and vocational education classes necessitated the fifth printing, which was accomplished early in 1960. Meanwhile the Committee had extended and strengthened the text, so that the present book, with 80 pages, 29 illustrations, and a careful index, is presented as an authoritative handbook for the teaching and the practice of first aid in industry, particularly the huge and scattered segment of it that is the multiple of the small plant. The professional qualifications and experience of the Committee members, and the care and pains they have taken in the long development of this work, well sustain the authority with which they clothe its presentation.

Prosthetic Principles: Above Knee Amputations

ANDERSON, BRAY and HENNESSY (Springfield, Illinois: Charles C. Thomas, 1960). 331 pp.

Because it is geared to the prosthetist, much of the material presented in this book is elementary as far as the physician is concerned, but clear and concise for the uninitiated. An excellent section, most of which also appears in *Clinical Prosthetics,* concerns itself with a detailed analysis of gait. This section deserves serious study by any physician concerned with the care of amputees.

The greatest part of the book is devoted to the fitting and fabrication of the above knee prosthesis. Each step is clearly described and well illustrated. About 17 pages are devoted to the principles of biomechanics which is defined as that science which deals with the forces that act on and in the human body. Knowledge of biomechanics both in the normal and in the amputee provides the student with a sound basis for understanding the problems of prosthetic prescription, fabrication and training.

This book is highly recommended for the practicing prosthetist. Physicians with a special interest in amputee work will find many pages of this book worth studying.

—A. A. RODRIQUEZ, M.D.

INDUSTRIAL MEDICINE AND SURGERY

ESTABLISHED 1932

Editor, CAREY P. McCORD, M.D.

Managing Editor, JOHN A. LAWRENCE

Consultant Editors:
CHARLES E. DUTCHESS, M.D.
CHESTER C. GUY, M.D.
SEWARD E. MILLER, M.D.

Abstracts:
MARVIN D. HENRY, M.D.
JOHN M. JOHNSTON, M.D.
ROBERT COLLIER PAGE, M.D., F.A.C.P., M.R.C.H.
ARTHUR A. RODRIQUEZ, M.D.
JOHN H. SCHNEEWIND, M.D.
W. A. WICHOWSKI, M.D.

Associate Editors:
L. E. HAMLIN, M.D.
EDWARD C. HOLMBLAD, M.D., F.A.C.S.
RAYMOND HOUSEHOLDER, M.D.
RUTHERFORD T. JOHNSTONE, M.D.
HEDWIG S. KUHN, M.D.
CHRISTOPHER LEGGO, M.D.
M. N. NEWQUIST, M.D.
OSCAR A. SANDER, M.D.
GERRIT W. H. SCHEPERS, M.D.
JAMES K. STACK, M.D., F.A.C.S.
ROBERT M. WATROUS, M.D.
REX H. WILSON, M.D.
EDWARD R. ASTON, D.D.S.
WARREN A. COOK, A.B.
W. B. DEICHMANN, Ph.D., F.R.S., MED.
HENRY FIELD SMYTH, Jr., Ph.D.
WILLIAM P. YANT, D.SC.

Overseas:
DR. AXEL AHLMARK, Sweden
PROF. DR. MED. E. W. BAADER, Germany
DR. H. P. DASTUR, India
DR. JOHN F. EUSTACE, Ireland
DR. DOUGLAS GORDON, Australia
DR. H. O. HOFMEYR, Union of South Africa
DR. BRANKO KESIĆ, Yugoslavia
DR. ARTHUR ERNESTO MONIZ, Portugal
PROF. DR. GIOVANNI PANCHERI, Italy
PROF. DR. JOSE PEDRO REGGI, Argentina
DR. RAFAEL PENALVER, Cuba
DR. A. SALMONT, France
DR. PERTTI SUMARI, Finland
DR. MASASHI SUZUKI, Japan
DR. A. R. THOMPSON, England
DR. A. UYTDENHOEF, Belgium

June, 1960

VOLUME XXIX

NUMBER 6

THE THIRTEENTH INTERNATIONAL CONGRESS ON OCCUPATIONAL HEALTH

New York, July 25-29, 1960

On July 25, 1960, the 13th International Congress on Occupational Health will convene in New York City under the President of the Permanent Commission, Dr. Sven Forssman of Sweden. The past four Congresses assembled in Helsinki, Naples, Lisbon, and London.

The hosts for the forthcoming Congress are the industrial physicians, industrial nurses, and industrial hygienists of the United States and Canada. Symbolically, however, the host will be the Organization Committee under the chairmanship of Dr. Leo Wade of New York City. The United States delegation to the International Congress has distinct duties as hosts, as have all organizations devoted to occupational health in the United States and Canada. But no one of those groups is the official host — the official host is you.

The Organization Committee, with its numerous subcommittees, has worked prodigiously and accomplished mightily. Thousands of items of correspondence, forms, reservations, and programs have been mailed. Thousands of details of arrangements have been achieved. Unstinted credit and gratitude for a successful congress heartily will be accorded the Organization Committee, since all such and more has been earned. But the final touch of success for the Congress will be determined by the hundreds of Americans who attend.

Representatives from at least 43 nations will attend this Congress. The majority will never have been in this country previously. These guests will expect much; perhaps more than may be fulfilled. In return for the regal entertainment that has characterized previous congresses, these visitors deserve all that we may provide.

The first duty of those thousands who are the official hosts is to attend, to be on hand to mingle, to participate. A second duty, scorning all cliques, and any form of aloofness, is to pour out the warmth that is expected of all who are Americans. All delegates represent the aristocracy of occupational health in their many countries. All groups will be found to be grand lots of human beings. This Thirteenth International Congress is indeed a scientific Congress, but, more important, this is a human Congress. There will be endless opportunities to display cordiality and to render hospitality, for there will be strangers and guests in our midst.

IN EMOTIONALLY PROJECTED
SMOOTH-MUSCLE SPASM...

Prompt, Profound Protection...at both ends of the vagus

PRO-BANTHĪNE® *with* DARTAL®

Professional reliance on the therapeutic proficiency of Pro-Banthīne in functional gastrointestinal disorders has made it the most widely prescribed anticholinergic.

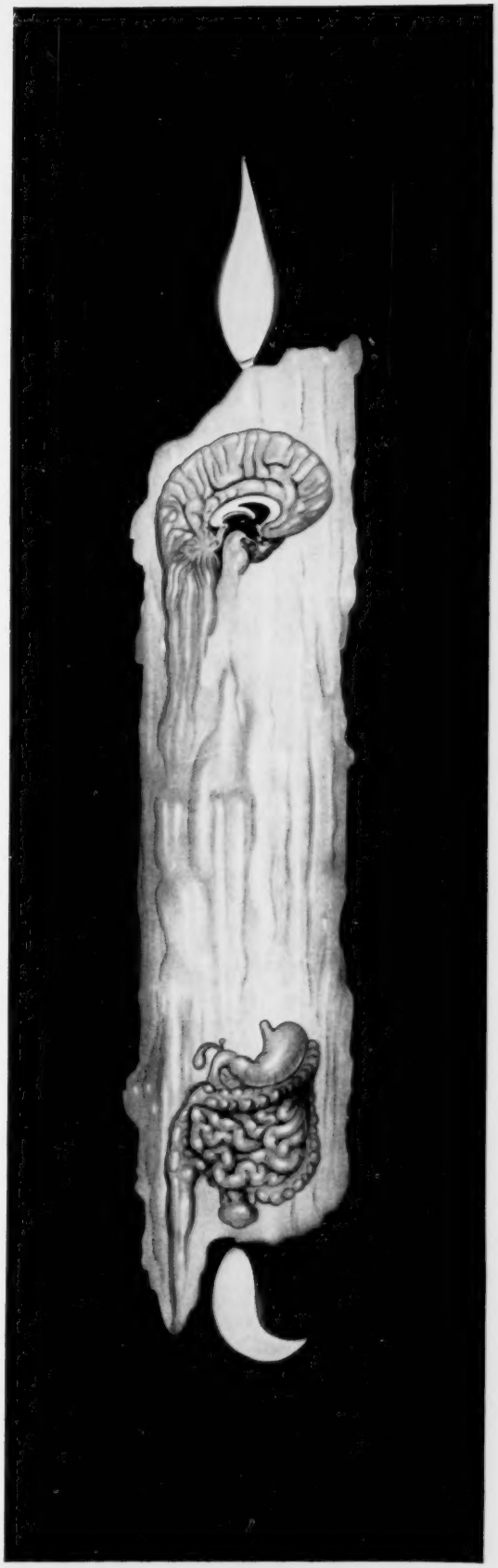
The consistent relief of emotional tensions afforded by Dartal makes this well-tolerated tranquilizer a rational choice to support the antispasmodic action of Pro-Banthīne in emotionally influenced smooth-muscle spasm.

These two reliable agents combined as Pro-Banthīne with Dartal consistently control both disturbed mood and disordered motility when emotional disturbances project themselves through the vagus to provoke such gastrointestinal dysfunctions as gastritis, pylorospasm, peptic ulcer, spastic colon or biliary dyskinesia.

USUAL ADULT DOSAGE:
One tablet three times a day.

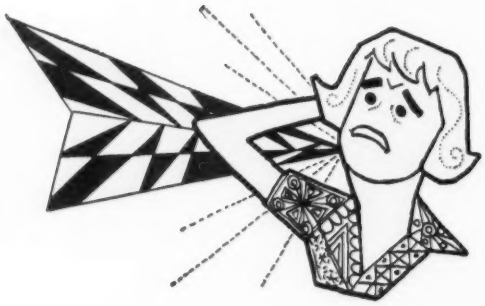
SUPPLIED as aqua-colored, compression-coated tablets containing 15 mg. of Pro-Banthīne (brand of propantheline bromide) and 5 mg. of Dartal (brand of thiopropazate dihydrochloride).

G. D. SEARLE & CO.
Chicago 80, Illinois
Research in the Service of Medicine



1. ANACIN is superior to aspirin or buffered aspirin...

2. In the management of tension headache



3. ANACIN possesses selective sedative action that eases tension, pressure as well as the pain

ANACIN®
ANALGESIC TABLETS

For a better 'total' effect in pain-relief

WHITEHALL LABORATORIES, NEW YORK, N.Y.

Here's Mote in Your Eye

A good cry may be just what the doctor ordered when dirt blows in your eye.

Tears are the quickest, easiest remedy for removing a small particle that lodges in the eye, according to John W. Smillie, M.D., Assistant Professor of Ophthalmology at The University of Michigan Medical Center.

"Emergency first aid is often the most important part of treating a potential eye infection," he points out, "and simple precautions can be applied on the spot."

First, wait a few minutes for tears to come naturally. The flooding action may wash away the foreign matter in an instant.

If the speck is still there, grasp your upper eyelashes and pull them down over the lower lid. The lower lashes serve as a brush to sweep out particles hidden under the upper lid.

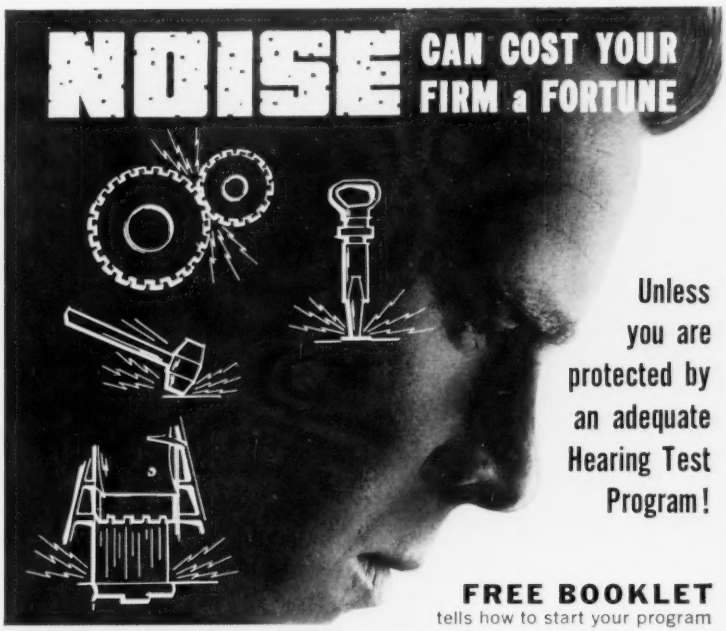
"Above all, don't rub or press on the eyeball," cautions the U-M doctor. This is often the hardest rule to follow when something's stinging in your eye. But rubbing causes pressure, he says, and a danger of imbedding the particle into the fiber of your eyeball.

If these remedies don't work, Dr. Smillie advises that you consult a physician "because even the most trivial eye abrasions can lead to serious complications."

Infections like a sty or pink eye can be treated at home with warm compresses, using ordinary warm tap water and washcloths. A physician may prescribe further medication, he says, and it is wise to consult him.

A black eye, on the other hand, is best relieved with a cold compress to reduce swelling. Then he suggests using hot compresses to help get rid of the discoloration.

Eye cuts — from home or


NOISE CAN COST YOUR FIRM a FORTUNE
Unless you are protected by an adequate Hearing Test Program!
FREE BOOKLET
tells how to start your program

FROM THE ACOUSTIC PRODUCTS DIVISION OF

MAICO

Electronics, Inc.

21 No. 3rd St. • Room 139F Minneapolis, Minn.

WRITE FOR FREE DATA

NAME _____

COMPANY _____

ADDRESS _____

CITY _____ STATE _____ (ZONE)

OUR 64th YEAR

WOODWARD MEDICAL PERSONNEL BUREAU

FORMERLY AZNOES

185 N. Wabash-Chicago, Ill.

Founders of the counseling service to the medical profession, serving medicine with distinction over half a century.

Telephone: RAndolph 6-5682

POSITIONS WANTED

Medical Director: 6 yrs, Asst Med Dir, important chemical co; since 1953, successful private genl pract; prefers indus med with regular hours; Atlantic seaboard, Ohio, Pa; early 40's.

Medical Director: Dipl, Int Med; 3 yrs, priv genl pract; 10 yrs, priv pract, Int Med; 4 yrs, Asst Med Dir, lge automotive indus; seeks lrge multi-plant indus; any location.

Medical Director: MPH; Cert'd, Occupational Med; 3 yrs, priv pract; 4 yrs, Capt, Med Corp; sevl yrs, Assoc Exam'g Phys, Hlth Dept; 2 yrs, lge corp, Med Dir; seeks Eastern locality—prefs N.Y., N.J., or Conn.

Medical Director: Fellow, Indus Med Assn; previous work in Surg & Toxicology; presently, staff phy of leading co; now seeks med dirshp or east med dirshp, indus med; Age 37.

Medical Director: 9 yrs, priv pract; 3½ yrs, med ofc, US Army; 6½ yrs, Med Dir, lge nationally famous co; seeks position w/advancement possibilities; any location; middle 40's.

Medical Director or Assistant: Eligible, Occupational Med; 4 yrs, private group; 4 yrs, exam'g & staff phys, lge hsp; seeks Denver, NY or Midwest areas; Age 33.

Internist: Board Elig (cardio); trnd univ hosp; interested Indus med as career; seeks further exper in adm or clinical capacity; 2 yrs, indus exper as staff phy & district chief; grad Hahneman; early 30's; prefers Pa, N.J., N.Y., Del or eastern seaboard.

Staff Physician: 2 yrs, Director, Blood & Plasma entr; three and one-half yrs private general practice; 3 yrs, Med Ofc, USAF; seeks Indus Med in SE, SW or California.

Staff Physician: Grad, Rush Med; 13 yrs, very successful private general & surgical pract; AAGP; sevl yrs, flight surgeon, USAF; prefers indus med with regular hours; no locality preference; middle 40's.

Staff Physician: 3 yrs, medical staff member, large SW corp; leaving because of family illness—seeks Indus post, Calif, SW or SE company; immediately available.

Staff Physician: A.B., Northern Univ; three years, private, general practice; prefers Indus postion; West or Northwest; Age 33.

POSITIONS OPEN

Internist: well trained, w/demonstrated ability to direct, full time, expanding Occup hlth prog nr headquarter ofc of important co, leader in its field; soft goods indus; oppor med schl tchg; lge city; E.

Associate Physician: Industrial med diagnostic dept, hdqtr ofc impor co; shld be internist, w/gd exper, prev med; NYC.

Medical Director: Internist or exper'd, indus med; automotive indus; substantial sal, fringes, retirement prog equivalent $18-20,000 from priv pract; Bay area, Cal.

Industrial Physician: w/Internal medicine background; lge, important co; $14,000; university city, W.

Industrial Physician: staff; recent grad; major co; 14,000 emplys; toxicology; $12,000 plus priv pract; city 500,000; med schl; MW.

Industrial Physician: July 1st; Assn, outstandg dept; apprvd IMA prog plus apprvd in-plant res, occ-med; lge co; to $18,000; 40 hrs; reqs lic, Wash State.

Industrial Physician: Machinery & chem corp; about 200 emplys; self-sustaining hsp; $12,-$15,000; small town, Midwest.

General Practitioners: large American co; construction project outside US; single, US citizen only; about $16,000; 1 yr contract.

IF IN NEED OF MEDICAL PERSONNEL, WRITE US SO WE MAY PREPARE AN INDIVIDUAL SURVEY FOR YOU

We offer you our best endeavor—our integrity—our 64 year record of effective placement achievement

STRICTLY CONFIDENTIAL

auto accidents — are more serious and should be treated immediately by an ophthalmologist. Dr. Smillie says the best thing to do is cover the injured eye with a soft clean cloth to prevent infection.

Catastrophe

Catastrophes — accidents in which five or more persons lost their lives — killed more than 1,400 persons in 1959, or about 150 fewer than in the preceding year, it is reported by statisticians of the Metropolitan Life Insurance Company.

The more favorable record for 1959 reflects the decreased number of fatal injuries in major catastrophes — accidents in which at least 25 people are killed — the statisticians point out. The loss of life in such catastrophes dropped from nearly 430 in 1958 to about 175 last year, an appreciably lower toll than in any year since 1941.

Four of the five major catastrophes in 1959 involved civil aviation. In February, a scheduled plane plunged into the East River in New York City, killing 65; another plane disintegrated in the air in a thunderstorm near Baltimore in May, taking 31 lives; in October a crash near Charlottesville, Va., killed 26; and 25 died when a scheduled plane rammed a mountain near Williamsport, Pa., on December first. The fifth major catastrophe was a series of earthquakes which took place on August 17 in the area of West Yellowstone, Mont., claiming about 29 victims.

Motor vehicle accidents were responsible for about two fifths of the lives lost in all catastrophes last year. Fires and explosions accounted for about one fourth and civil aviation for somewhat less than one fifth of such deaths. The remaining fatalities were very largely due to natural catastrophes and to accidents involving military aviation, water transportation, and railroads.

Now in a
PROFESSIONAL PACK
BARD-PARKER
STERILE BLADES
Individual, puncture-resistant, reinforced foil packages . . . that can be autoclaved if desired — help protect these traditionally sharper carbon steel blades. Handy new Professional Pack holds 6 Sterile Blade packages of a size.
And, time-tested B-P RIB-BACK Blades are also available in the RACK-PACK package or in rust-resistant wrappers holding 6 blades of a size.
It's Sharp
B-P • IT'S SHARP • RACK-PACK • RIB-BACK are trademarks
Ask your dealer
B-P
BARD-PARKER COMPANY, INC.
DANBURY, CONNECTICUT
A DIVISION OF BECTON, DICKINSON AND COMPANY

The Eyes Have It

More than half the people in this country have some kind of visual defect. Only 43.5% of Americans tested in one survey cited by *Patterns* had normal vision — that is, 20-30 vision or better in both eyes. Almost one in 10 were found to have the use of only one eye.

The impression this survey gives of a nation afflicted with weak eyes is strengthened by other figures reported in the publication. One out of every two Americans — an estimated total of 83,500,000 — wears either eyeglasses or contact lenses.

Further statistics show there are 960,000 blind persons in the country and an additional 2,064,000 with serious visual impairments. Together, blindness and poor vision rank fourth in the list of disabilities in the United States, outranked only by impairments of hearing; of the limbs, back and trunk; and disorders of the lower extremities.

When it comes to a question of the sexes, men do better visually than women. In one study of over 100,000 persons, it was found that in the 30-35 age group, approximately 42% of men suffered from defective vision, compared to about 48% of the women. In the over-40 age group, the difference was even

more marked — about 10% to 12% higher for women than for men.

Marked differences also are found in breakdowns by occupation. Clerical and administrative workers top the list of visually defective persons in one study with 51% requiring glasses. At the other end of the list on the same survey are drivers of mobile equipment, with only 27.2% wearing glasses.

Industrial workers as a group seem to be particularly vulnerable to eye trouble, not so much because of any inherent weakness as because of the high proportion of eye injuries that occur in industry.

One thousand eye injuries occur every day of industrial operation. At least 90% of these are needless and, aside from the physical and economic suffering of the victim and his family, are highly costly to industry.

Most of these injuries could be avoided, as evidenced by the outstanding success of mandatory eye safety programs which certain industries have instituted. One large Ohio plant which once had 500 to 600 eye injuries a month now has none. Another plant, in the East, has saved $1,000,000 in compensation costs.

—From *Patterns of Disease*, April, 1960

Take It Off

Women withstand the effects of overweight better than men do, but excess weight adversely affects their health and longevity, it is reported by statisticians of the Metropolitan Life Insurance Company. For the same degree of overweight, the excess mortality among women is less than it is among men, according to information which has recently become available from a study by the Society of Actuaries. Thus, among women aged 15-39 who were 20% overweight at the time they obtained ordinary insurance, the mortality was 15% above that for standard female risks; the comparable group of men had a mortality 24% higher than that for all standard male risks.

Despite their advantage over men, there is no reason for overweight women to become complacent, the statisticians point out. Overweight women in every age period have a higher mortality than those whose weight is close to average, the excess mortality increasing with advance in age and degree of overweight.

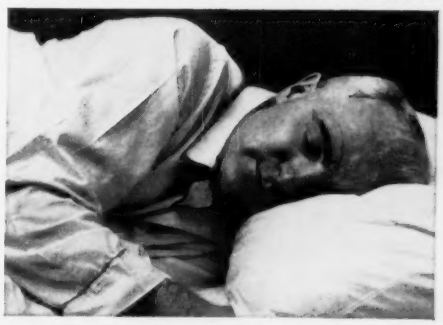
The disadvantage of overweight in women shows up even more distinctly when their mortality is compared with that of moderately underweight women. On this basis, the excess mortality was 30% for women who were 10% overweight when this insurance was issued, and 40% for those who were 20% or more overweight.

Heart disease ranks first among the causes of death for the overweight women, the death rate being 50% above that for standard female risks. Other diseases with much higher death rates among overweight women are cancer, diabetes, pneumonia

for the uncomfortable patient for the distraught patient for the overtired patient

relaxing, restful sleep

without barbiturates, bromides or narcotics



sominex®

THE SAFE SOMNIFACIENT

SOMINEX contains no barbiturates, bromides or narcotics. It is designed specifically as a bedtime sedative, and should not be used as a daytime tranquilizer.

In SOMINEX, the safe sedative action of methapyrilene is enhanced by scopolamine and salicylamide. The total effect is one of safe sedation without hang-over or danger of habituation. No prescription is required.

Each SOMINEX tablet provides: Methapyrilene HCl, 25 mg.; scopolamine aminoxide HBr, 0.25 mg.; salicylamide, 200 mg. *Dosage:* 2 tablets one-half hour before retiring. Some patients will require only one tablet. *Supplied:* vials of 18 tablets.

For a complimentary supply, please address your request to: Dept. SC, J. B. Williams, Inc., 711 Fifth Avenue, New York, N.Y.

and influenza, and diseases of the digestive system.

The splendid health record of women in the general population of the United States and Canada — among the best in the world — could be further improved if overweight women shed their excess weight. This, it is expected, would reduce mortality from those conditions which account for the excess mortality of overweights. Indeed, the control of conditions in which overweight plays a distinct role offers the best prospects for further substantial reduction in the already low death rate of women.

Health Makes Wealth

Your industry executives are rapidly expanding company-paid annual medical check-up programs, paying more attention to the famous old Disraeli adage, "health is the individual's greatest capital asset . . . without health one can neither work, nor earn."

How to set up a health examination program? Where? How much will it cost? These question can be answered very simply and an adequate plan will cost relatively little as measured against the potential benefits to executive and company.

The first step is to understand thoroughly that a *comprehensive* examination is a must. A simple chest thumping and urinalysis may create a false sense of security — hurt more than it helps. Recently, a leading business publication outlined the following minimum program:

Full medical history-family background, diseases, operations.

Head-to-toe examination, to reveal special trouble areas.

X-ray of the heart and lungs; also an electrocardiogram which charts the action of the heart (often omitted in less searching examinations).

Rectal examination, both digital and sigmoidoscope (the latter urged by the American Cancer Society as a wise safeguard).

Blood count to reveal anemia, nutritional disorders, infections; blood sugar test for diabetes; blood cholesterol test, as a possible safeguard against certain heart and coronary blood vessel diseases, blood non-protein nitrogen test, bearing on kidney function, and blood uric acid test to detect "hidden" gout.

Urinalysis, both chemical and microscopic.

Eye examination.

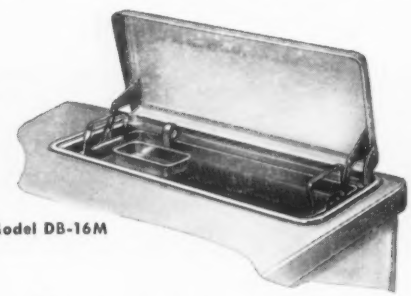
Concluding consultation and written report.

Additional x-ray surveys (gastrointestinal series, barium enema, gall bladder series)

NOW!
A Modern Centralized STERILE SUPPLY for Offices and Clinics . . .

Amsco

DOUBLE Cabinet Sterilizer . . .



Model DB-16M

Compact, convenient and easy to operate, the new Amsco Double Cabinet Sterilizer features a handsome double cabinet and utility drawer with ample storage space for instruments and supplies — *and a built-in automatically burn-out proof* **Office Instrument Sterilizer.** The cabinet is also available with a solid top without the recessed boiling-type sterilizer.

The large formica counter provides ample room for work space, while the roomy cabinet and drawers are safe for storage of sterile supplies . . . ready for instant use. The efficient, recessed type A-416S non-pressure sterilizer is fabricated entirely of stainless steel, insulated construction with two trays — one for instruments, the other for needles.

One of the ten popular colors available with the DB-16M will blend perfectly with your office decor. A single cabinet with the same mechanical features is also available.

Amsco's large Office Pressure-Steam Sterilizer, 613-R Dynaclave, or 8" Square Autoclave, Cat. No. 8816, can be conveniently located on work counter of the double cabinet.

AMERICAN STERILIZER
ERIE • PENNSYLVANIA

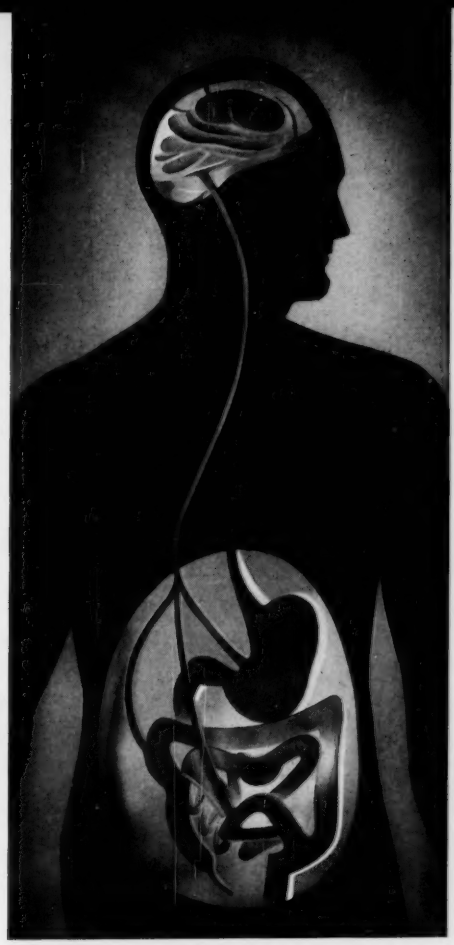
* *See the new Double Cabinet Sterilizers at your authorized Amsco dealer or write for Bulletin DC-404*

anticholinergic

KEEPS THE STOMACH FREE OF PAIN

tranquilizer

KEEPS THE MIND OFF THE STOMACH



Milpath acts quickly to suppress hypermotility, hypersecretion, pain and spasm, and to allay anxiety and tension with minimal side effects.

AVAILABLE IN TWO POTENCIES:

Milpath-400 – Yellow, scored tablets of 400 mg. Miltown (meprobamate) and 25 mg. tridihexethyl chloride. Bottle of 50.

Dosage: 1 tablet t.i.d. at mealtime and 2 at bedtime.

Milpath-200 – Yellow, coated tablets of 200 mg. Miltown (meprobamate) and 25 mg. tridihexethyl chloride. Bottle of 50.

Dosage: 1 or 2 tablets t.i.d. at mealtime and 2 at bedtime.

Milpath®

®Miltown + *anticholinergic*

WALLACE LABORATORIES *New Brunswick, N. J.*

should be made if there's a complaint of frequent heartburn, "nervous stomach," poor appetite, diarrhea, or constipation.

In addition, a check on emotional health may be required if there is evidence of excessive tension and stress. Ironically, one indication of the need for this kind of inquiry is an unreasonable fear of a standard physical examination.

Nowadays, a program like this can be arranged in almost any part of the country with a clinic which specializes in check-up plans.

Improvements in patient handling technique are one important reason for the marked increase in interest in annual check-ups. More executives are willing to go for an exam when they do not feel "pushed around" as is often the case. Disraeli was a bright old gentleman, and the industry will avoid unnecessary loss of key personnel through adherence to his philosophy and institution of health check-up plans.

—From *Best's Insurance News.*

OHI Certification

A medical program for employees that detects health problems before they become serious has won for British Columbia Electric recognition from the medical profession.

The coveted "Certificate of Health Maintenance" was presented recently to Dr. Harry L. Purdy, BCE executive vice-president, by Dr. Russell Birrell, of Toronto, on behalf of the Occupational Health Institute at the third annual Occupational Health Conference in Hotel Vancouver.

Only one other firm in British Columbia, Consolidated Mining and Smelting Ltd., of Trail, had been awarded the certificate.

The health service provided for employees by B.C. Electric is an excellent example of an enlightened management skillfully providing preventive medical service, Dr. Birrell stated. "In examining this service for certification, we found that the facilities and staff, headed by Dr. John Nelson, far exceeded the standards."

"But what impressed us most was the way in which employees have made use of these facilities.

"This indicates how conscientious and skillful a job of health education has been carried out and provides a lesson not only for other employing organizations but for communities.

"This service means the BCE employees who take advantage of the company's medical facilities receive early warning of heart disease, lung cancer, tuberculosis and many other illnesses which can be treated at early stages but which might become hopeless if detection facilities were not available."

NEW CAMP SACRO-ILIAC SUPPORTS

help steady the pelvis and offer excellent support to the sacro-iliac region and lower abdomen

Camp's new women's sacro-iliac supports firmly encircle the bony pelvis between crests of the ilia and trochanter, thus increasing the stability of the pelvic girdle and diminishing the rotary movements of the sacro-iliac joints. Both back and abdomen receive splendid support.

These sacro-iliac garments also perform the additional function of holding the stomach in a position helpful in medium severe cases of gastroptosis or nephroptosis. They are effective, too, for patients requiring support to weakened abdominal walls after a major operation.

The slim, trim modern lines modern women want in a foundation garment are incorporated in the design without sacrificing medical effectiveness.

These new Camp garments include such features as —

- a new band of support which works with boned front and back panels
- cable knit, strong elastic, side panels
- unique, quick, inconspicuous functional adjustment
- thin-design hose supporters
- skirt or semi-groin lengths in tea rose or white
- zipper closing.

S. H. CAMP & COMPANY, JACKSON, MICHIGAN

S. H. Camp & Company of Canada, Ltd., Trenton, Ontario

CAMP

CLINICAL HAND WASHING TESTS PROVE

CREME HAND CLEANER HAS DEFINITE BACTERIOSTATIC PROPERTIES

By reducing the number of bacteria and fungi accumulating on workers' hands, the probability of skin irritations and industrial dermatitis is diminished. In a recent clinical test,* subjects representing a wide range of occupations used Go-Jo Creme Hand Cleaner for twelve days. Upon comparing skin bacterial counts, it was found that the regular use of Go-Jo Creme Hand Cleaner was accompanied by a statistically significant reduction in the numbers of bacteria and fungi. The reduction was greater than 99% of the original count.

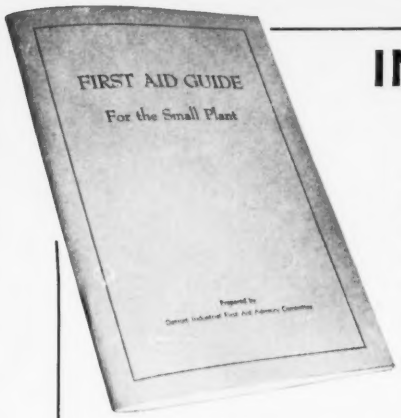
*Price Hand Washing Test (Jour. Amer. Med. Assoc. 111,1993-96,1938), as performed by an independent microbiological testing laboratory (name on request).

LET Go-Jo CREME HAND CLEANER WORK IN YOUR PLANT TO HELP PREVENT INDUSTRIAL DERMATITIS.

FOR FURTHER INFORMATION WRITE: DEPT. IMS6

GOJER, INC. Box 991 Akron 9, Ohio

MANUFACTURERS OF *go-jo* PRODUCTS 2231



INDUSTRIAL FIRST AID GUIDE

THIS 80-page Industrial First Aid Guide, expertly written, fully illustrated, completely indexed, tells what it is proper to do, and how to do it quickly and effectively, and — equally important — what **not** to do. It was prepared by Industrial Physicians and Surgeons and Industrial Hygienists* widely experienced in the requirements of Industrial First Aid. Where professional care is not at hand, it provides for the interval until such care can be obtained.

Chapters

Medical Care ● First Aid Kits ● First Aid Room ● Records ● General First Aid ● Shock ● Wounds ● Bandaging ● Special Wounds ● Artificial Respiration ● Fractures ● Transportation ● Dermatitis ● Burns ● Common Medical Emergencies ● Miscellaneous Medical Conditions ● Ill Effects of Excessive Heat ● Poisons ● Deaths ● First Aid in Civil Disaster.

80 pages; 29 illustrations.

Per Copy, 75 cents.

For quantity prices — Address:

First Aid Guide
Box 241, Winnetka, Illinois.

*First Aid Advisory Committee

A.H. WHITTAKER, M. D., Past-President, Industrial Medical Association, Chairman.

MARTIN P. BRUTON, M.D., Assistant Corporate Medical Director, Chrysler Corporation.

JOHN A. COWAN, M.D., Medical Consultant, Division of Occupational Health, Michigan Department of Health.

WILLIAM G. FREDRICK, Sc.D., Director, Bureau of Industrial Hygiene, Detroit Department of Health.

E. A. IRVIN, M.D., Medical Director, Ford Motor Company.

MARION W. JOCE, M.D., Corporate Medical Director, Chrysler Corporation.

ESTHER W. KILMER, R.N., Industrial Nursing Consultant, Detroit Department of Health.

D. W. LINDNER, M.D., Clinical Instructor in Neurosurgery, College of Medicine, Wayne State University.

EARL P. LUTZ, M.D., Associate Medical Director, General Motors Corporation.

PAUL T. SALCHOW, M.D., Detroit Department of Health.

HOMER M. SMATHERS, M.D., Senior Instructor in Clinical Surgery, College of Medicine, Wayne State University.

S. D. STEINER, M.D., Medical Director, General Motors Corporation.

W. GEORGE WIBBY, Director of Safety Services, American Red Cross, Detroit Chapter.

SAMUEL W. WICKHAM, Chief Inspector, Michigan Department of Labor.

massages pain away in musculoskeletal involvements[1]

GER-O-FOAM

(aerosol foam)

relieves pain, spasm; improves function increases tolerance to exercise

GER-O-FOAM's exclusive formula provides for the first time deeply absorbed analgesic-anesthetic agents in aerosol form — to permeate and anesthetize sensory nerve endings.

Relief in minutes, lasting for hours in . . . rheumatoid arthritis, osteoarthritis, muscle sprain, fibromyositis, low back pain . . . even in chronic intractable cases.

GER-O-FOAM combines: Methyl salicylate 30%, benzocaine 3%, in a neutralized emulsion base, permitting fast penetration through the stratum corneum.

Sample and reprint from

GERIATRIC PHARMACEUTICAL CORP.
Bellerose, New York

Pioneers in Geriatric Research

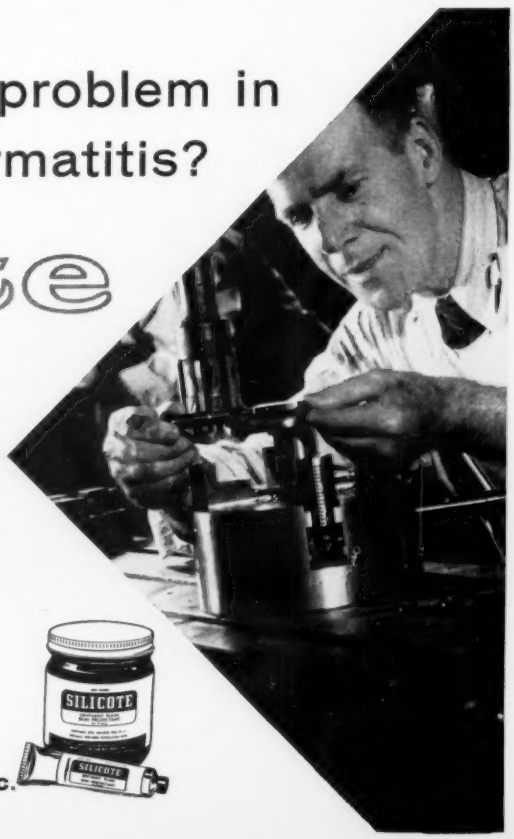
1. Gordon, E. E. and Haas, A.: Industrial Medicine & Surgery 28:217, 1959.

---

do you have a problem in industrial dermatitis?

Silicote

SILICONE SKIN PROTECTANT



Silicote provides an effective barrier against a wide range of skin irritants. It adheres to the skin, is not easily removed, and, when properly applied, can not be seen nor felt on the skin, permitting workers performing delicate tasks to stay on the job.

PROTECTS AGAINST

—water, acids, alkalies, foods, organic materials, resins, cement, tanning solutions, inks, polishes, woods, glue, coolants, soluble cutting oils, rubber, plastics, synthetics, dyes, metals, paints, lacquers, thinners, etc.

IN TREATMENT

of contact dermatoses. Silicote clears up many cases not responsive to other therapy.

Silicote is the only preparation containing 30% silicones (dimethicone) in specially prepared petrolatum base. Silicote is non-occlusive.

May we work with you in your particular problem? Send for clinical supply.

ARNAR-STONE LABORATORIES, INC.

225 E. PROSPECT AVE., MOUNT PROSPECT, ILLINOIS

balanced weave
assures
uniform
support

NOW...BETTER THAN EVER BECAUSE

B-D

a scientifically determined ratio of warp (lengthwise) to woof (cross) threads in every ACE Bandage provides a pressure pattern that—

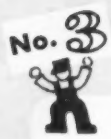
- guarantees even and controlled stretch
- insures firmness under tension
- prevents bunching
- minimizes possibility of vein constriction

ACE

RUBBER ELASTIC BANDAGE

BECTON, DICKINSON AND COMPANY
RUTHERFORD, NEW JERSEY

B-D AND ACE ARE REGISTERED TRADEMARKS. 77760



in a series of washroom survey comments from "SBS soap counselors"

We showed them how to clean - disinfect - sanitize - deodorize in one money-saving operation!

Edward T. McNamara, Jr.

SBS soap counselor, has helped many plants in the Southwest improve their wash-up and clean-up programs. Here's an excerpt from his account of a survey at a synthetic rubber plant:

"We found washroom housekeeping costs way up . . . for the usual reasons. Maintenance men were using slow-acting scrub soaps, scouring powder and disinfectants. These single-purpose products required lots of elbow grease and needless duplication of effort.

"Plant Management was pleasantly surprised when we showed them how easily a single product, new SBS 50 handles *ALL* of these washroom chores *in less than half the time!*" SBS 50 cleans - sanitizes - disinfects - deodorizes in *1 economical operation!* Just one swish of a mop or washcloth with SBS 50 cleans soils from walls, floors, fixtures — all the places where germs breed. No scrub, no rinse, no waste motion! SBS 50 is especially formulated to simplify industry's housekeeping chores. Its effective 4-way action reduces maintenance costs.

Money-saving suggestions like this *begin* with OPERATION PINPOINT—a thought-provoking presentation filled with facts about skin hygiene and washroom maintenance. The SBS soap counselor serving your area can pinpoint the right soap to do every skin cleansing job best and at lowest cost in your plant. Let him show you OPERATION PINPOINT . . . just call your nearest SBS office, collect.

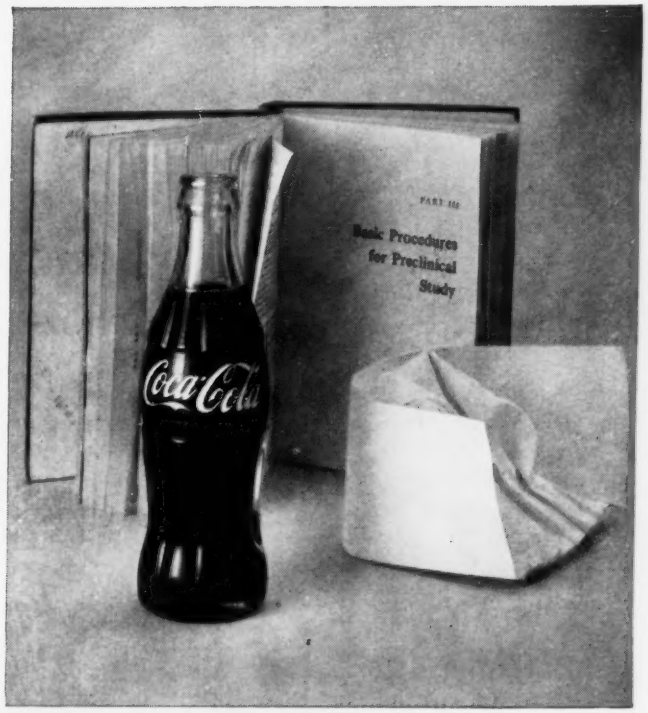
the washword of industry

SBS sugar beet products co.

SAGINAW, MICH. • Los Angeles, Calif. • Newark, N. J.
CANADA: Chemical By-Products, Ltd., Toronto, Ontario

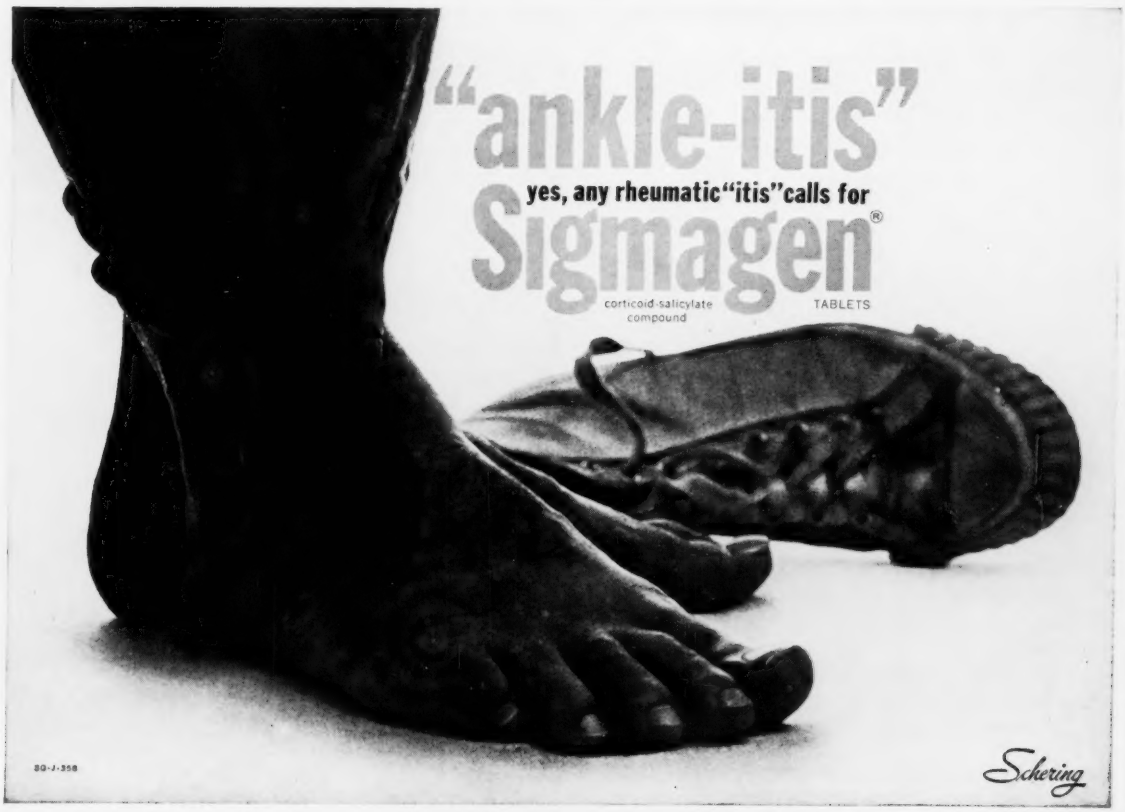
Index of Advertisers

American Sterilizer Co. . . . 38
American Tobacco Co. . . . 26
Armour Pharmaceutical Co. . . . . 14, 3rd cover
Arnar-Stone Laboratories . . 42
Bard-Parker Co., Inc. 36
Becton, Dickinson & Co. 8, 43
Bristol-Myers Co. 7
Burdick Corp. 46
Camp & Co., S. H. 40
Carbisulphoil Co. 46
Ciba Pharmaceutical Products, Inc. 30
Coca Cola Co. 45
Desitin Chemical Co. 23
Dome Chemicals, Inc. . . . 20
Eaton Laboratories 10, 11
Geriatric Pharmaceutical Corp. 42
Gojer, Inc. 41
Ille Electric Corp. 46
Industrial First Aid Guide 41
Lilly & Co., Eli 32
McNeil Laboratories, Inc. 31
Maico Electronics, Inc. . . 35
Merck Sharp & Dohme . . 16
National Dairy Council . . . 19
Riker Laboratories, Inc. 2nd cover
Robins, Co., Inc., A. H. . . . 9
Roerig & Co., J. B. 2
Schering Corp. 1, 45
Searle & Co., G. D. 33
Squibb & Sons, E. R. . . . 18, 25
Sugar Beet Products Co. . . 44
Syntex Chemical Co., Inc. 22
Tailby-Nason Co., Inc. 5
Upjohn Co. 28, 29
Wallace Laboratories 12, 13, 39, 4th cover
Warner-Chilcott Laboratories, Inc. 15
White Laboratories, Inc. . . 27
Whitehall Laboratories 24, 34
Williams, Inc., J. D. 37
Winthrop Laboratories . . . 17
Woodward Medical Personnel Bureau 35
Wyeth Laboratories, Inc. . . 21



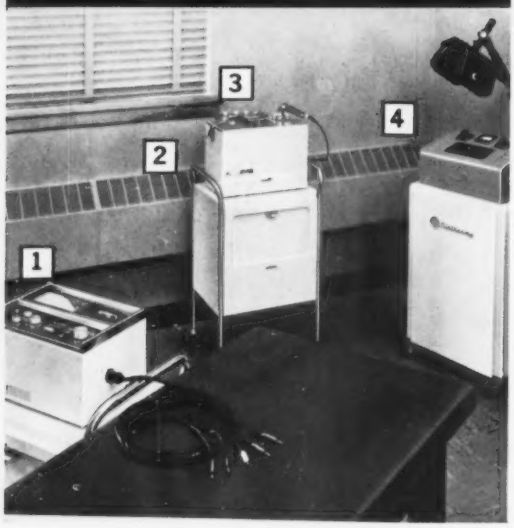
When too many tasks
seem to crowd
the unyielding hours,
a welcome
"pause that refreshes"
with ice-cold Coca-Cola
often puts things
into manageable order.

DRINK
Coca-Cola
REG. U.S. PAT. OFF.



Burdick

electronic aids for diagnosis and treatment



With the advances and new technics in electronics as applied to medicine, efficient and practical equipment is now available for general office use—

1 **EK-III Dual-Speed ELECTROCARDIOGRAPH** — Lightweight, portable, accurate, simple to operate. 25 mm.- or 50 mm.-per-second speeds.

2 **UT-400 PULSED ULTRASONIC UNIT** — Continuous or pulsed energy. Compact, portable, six sq. cm. radiating area.

3 **MS-300 MUSCLE STIMULATOR** — Ideal for stimulation of innervated muscle tissue. Can be used in combination with the UT-400, as illustrated above.

4 **MF-49 SHORT WAVE DIATHERMY** — Versatile. Used with every type of diathermy electrode.

Complete information — including specifications and prices — on all Burdick electromedical apparatus is readily available from your local Burdick representative, or write directly to The Burdick Corporation, Milton, Wisconsin.

THE BURDICK CORPORATION
MILTON, WISCONSIN
Branch Offices:
New York • Chicago • Atlanta • Los Angeles
Dealers in all principal cities

FOILLE® FIRST AID *Spray* FOR BURNS

FOILLE — the antiseptic, analgesic dressing — is indicated for fast, effective relief from pain of burns, sunburn, cuts, wounds and abrasions. Large or small areas can be sprayed thoroughly to provide prompt patient comfort and eliminate painful swabbing. FOILLE fights infection and promotes healing.

CARBISULPHOIL COMPANY • DALLAS, TEXAS

NOW in 3-oz. and 10-oz. SPRAY . . .

ANTISEPTIC • ANALGESIC
FOILLE®

ORDER **FROM YOUR FIRST AID SUPPLIER**

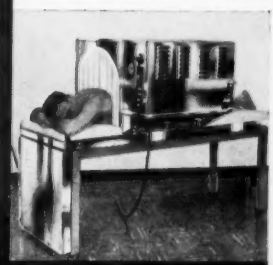
ILLE: A DISTINGUISHED NAME IN HYDRO- AND PHYSICAL THERAPY EQUIPMENT

←
HM-655
COMBINATION ARM, LEG or HIP UNIT
(Stationary)

For subaqua therapy. Stainless steel tank with electric turbine ejector and thermostatic mixing valve.

MA-105 →
MOISTAIRE HEAT THERAPY UNIT

Delivers temperature-controlled moist heat safely and effectively. Complete with stainless steel treatment hood, table, latex foam table pad, nylon moistureproof curtains and 4-quart filling can.



ILLE ELECTRIC CORPORATION
Reach Road, Williamsport, Pa.

Chymar ". . . hastens absorption of edema and blood in the tissues . . . acts as a remarkable anti-inflammatory agent."[1]

"The pain and swelling in all cases was remarkably decreased in from 24 to 48 hours."[2]

". . . edema, erythema and pain apparently were alleviated markedly and rapidly."[3]

1. Fullgrabe, E. A.: Ann. New York Acad. Sc. 68:192, 1957. 2. Jenkins, B. H.: J.M.A. Georgia 45:431, 1956. 3. Davis, O. F., et al.: Postgrad. Med. 26:719, 1959.

TRAUMA? CHYMAR®

Buccal / Aqueous / Oil

the superior anti-inflammatory enzyme *controls inflammation, swelling and pain*

CHYMAR BUCCAL—Crystallized chymotrypsin in a tablet formulated for buccal absorption. Bottles of 24 tablets. Enzymatic activity, 10,000 Armour Units per tablet.

CHYMAR AQUEOUS—Solution of crystallized chymotrypsin in sodium chloride injection for intramuscular use. Vials of 5 cc. Enzymatic activity, 5,000 Armour Units per cc.

CHYMAR—Suspension of crystallized chymotrypsin in oil for intramuscular injection. Vials of 5 cc. Enzymatic activity, 5,000 Armour Units per cc.

THE SYSTEMIC ROUTE TO FASTER HEALING AT ANY LOCATION

ARMOUR PHARMACEUTICAL COMPANY • KANKAKEE, ILLINOIS *Armour Means Protection* ®

© 1960, A. P. Co.

Proven

in over five years of clinical use and
more than 750 published clinical studies

Effective

for relief of anxiety and tension

Outstandingly Safe

- simple dosage schedule produces rapid, reliable tranquilization without unpredictable excitation
- no cumulative effects, thus no need for difficult dosage readjustments
- does not produce ataxia, change in appetite or libido
- does not produce depression, Parkinson-like symptoms, jaundice or agranulocytosis
- does not impair mental efficiency or normal behavior

Miltown®

meprobamate (Wallace)

Usual dosage: One or two 400 mg. tablets t.i.d.
Supplied: 400 mg. scored tablets, 200 mg. sugar-coated tablets; or as MEPROTABS®– 400 mg. *unmarked*, coated tablets.

WALLACE LABORATORIES / *New Brunswick, N. J.*

OM-2058

®TRADE-MARK